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☐ 1. Document ID: US 20020132225 A1

L1: Entry 1 of 4 File: PGPB Sep 19, 2002

PGPUB-DOCUMENT-NUMBER: 20020132225
PGPUB-FILING-TYPE: new
DOCUMENT-IDENTIFIER: US 20020132225 A1

TITLE: Compositions and methods for prolonging survival of chilled platelets

PUBLICATION-DATE: September 19, 2002

INVENTOR-INFORMATION:

NAME	CITY	STATE	COUNTRY	RULE-47
Stossel, Thomas P.	Belmont	MA	US	
Hartwig, John H.	Jamaica Plain	MA	US	
Wagner, Denisa D.	Wellesley	MA	US	

US-CL-CURRENT: 435/4; 435/7.2

Full | Title | Citation | Front | Review | Classification | Date | Reference | Sequences | Attachments | Claims | KWIC | Draw Desc | Image

☐ 2. Document ID: US 20020040008 A1

L1: Entry 2 of 4 File: PGPB Apr 4, 2002

PGPUB-DOCUMENT-NUMBER: 20020040008
PGPUB-FILING-TYPE: new
DOCUMENT-IDENTIFIER: US 20020040008 A1

TITLE: Method for treating and preventing atherosclerosis

PUBLICATION-DATE: April 4, 2002

INVENTOR-INFORMATION:

NAME	CITY	STATE	COUNTRY	RULE-47
Wagner, Denisa D.	Wellesley	MA	US	
Johnson, Robert C.	Sparta	NJ	US	

US-CL-CURRENT: 514/41; 424/130.1, 514/54, 514/8

Full | Title | Citation | Front | Review | Classification | Date | Reference | Sequences | Attachments | Claims | KWIC | Draw Desc | Image

☐ 3. Document ID: US 20020031508 A1

L1: Entry 3 of 4 File: PGPB Mar 14, 2002

PGPUB-DOCUMENT-NUMBER: 20020031508

PGPUB-FILING-TYPE: new
DOCUMENT-IDENTIFIER: US 20020031508 A1

TITLE: Methods for diagnosing and treating hemostatic disorders by modulating P-selectin activity

PUBLICATION-DATE: March 14, 2002

INVENTOR-INFORMATION:

NAME	CITY	STATE	COUNTRY	RULE-47
Wagner, Denisa D.	Wellesley	MA	US	
Andre, Patrick	Jamaica Plain	MA	US	
Hartwell, Daqing W.	Brookline	MA	US	
Hrachovinova, Ingrid	Jamaica Plain	MA	US	

US-CL-CURRENT: 424/94.63; 424/145.1, 514/12

Full | Title | Citation | Front | Review | Classification | Date | Reference | Sequences | Attachments | Claims | KWIC | Draw Desc | Image

☐ 4. Document ID: US 5807745 A

L1: Entry 4 of 4 File: USPT Sep 15, 1998

US-PAT-NO: 5807745
DOCUMENT-IDENTIFIER: US 5807745 A

TITLE: Method of inhibiting PADGEM-mediated or ELAM-1-mediated leukocyte adhesion using an inhibitor comprising a Le.sup.x core component

DATE-ISSUED: September 15, 1998

INVENTOR-INFORMATION:

NAME	CITY	STATE	ZIP CODE	COUNTRY
Furie; Bruce	Wellesley	MA		
Furie; Barbara C.	Wellesley	MA		
Larsen; Eric	Lebanon	NH		
Palabrica; Theresa	Quincy	MA		
Sajer; Susan A.	Brookline	MA		
Wagner; Denisa D.	Wellesley	MA		

US-CL-CURRENT: 435/375; 436/501, 436/63

Full | Title | Citation | Front | Review | Classification | Date | Reference | Sequences | Attachments | KWIC | Draw Desc | Image

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Term	Documents
WAGNER-DENISA$	0
WAGNER-DENISA-D.USPT,PGPB.	4
WAGNER-DENISA$.USPT,PGPB.	4
(WAGNER-DENISA$).USPT,PGPB.	4

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Search Results - Record(s) 1 through 10 of 24 returned.

☐ 1. 20020098563 . 02 Mar 01. 25 Jul 02. Novel core 2 béta-1,6-N-acetylglycosaminyltransferase gene. Korczak, Bozena, et al. 435/193; 435/320.1 435/325 435/69.1 536/23.2 C12N009/10 C07H021/04 C12N005/06.

☐ 2. 20020076833 . 01 Aug 01. 20 Jun 02. Analysis of biological samples utilizing a coated solid phase. Henry, Michael R., et al. 436/518; G01N033/543.

☐ 3. 20020061863 . 02 Nov 01. 23 May 02. Novel, specific inhibitors of acute and chronic inflammation. Uppugunduri, Srinivas. 514/49; 514/269 514/738 A61K031/7115 A61K031/513 A61K031/047.

☐ 4. 20020058034 . 12 Jul 01. 16 May 02. Inhibition of differentiation of cytotoxic T-cells by P-selectin ligand (PSGL) antagonists. Manjunath, Narasimhaswamy, et al. 424/144.1; 514/12 514/54 A61K039/395 A61K038/17 A61K031/726.

☐ 5. 20020045202 . 28 Feb 01. 18 Apr 02. Novel core 2 beta-1,6-N-acetylglycosaminyltransferase gene. Korczak, Bozena, et al. 435/15; 435/193 435/252.3 435/320.1 435/325 536/23.2 C12N009/10.

☐ 6. 20020040008 . 18 Jun 01. 04 Apr 02. Method for treating and preventing atherosclerosis. Wagner, Denisa D., et al. 514/41; 424/130.1 514/54 514/8 A61K039/395 A61K031/715 A61K038/16.

☐ 7. 20020037840 . 23 Mar 01. 28 Mar 02. Novel P-selectin glycoprotein ligand (PSGL-1) binding protein and uses therefor. Lorenz, Meike, et al. 514/8; 435/325 435/6 435/69.1 530/395 536/23.5 C12Q001/68 A61K038/16 C07H021/04 C12P021/02 C12N005/06.

☐ 8. 20020025923 . 18 May 01. 28 Feb 02. Novel selection ligands. Rosen, Steven D., et al. 514/8; 530/395 A61K038/17 C07K014/435.

☐ 9. 20010046970 . 22 Jun 01. 29 Nov 01. Inhibition of selectin binding. Nagy, Jon O., et al. 514/53; 514/54 A61K031/726 A61K031/715.

☐ 10. 20010036931 . 27 Apr 01. 01 Nov 01. Inhibition of cell-cell binding by lipid assemblies. Nagy, Jon O., et al. 514/53; A61K031/7016.

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Term	Documents
ATHEROSCLEROSIS.USPT,PGPB.	15316
ATHEROSCLEROSES.USPT,PGPB.	30
ARTERIOSCLERORIS.USPT,PGPB.	1
ARTERIOSCLERORI	0
PSGL$	0
PSGL.USPT,PGPB.	84
PSGLB1.USPT,PGPB.	1
"PSGLB1/MIGG.SUB.2B".USPT,PGPB.	1
PSGLITSPNW.USPT,PGPB.	2
PSGLL.USPT,PGPB.	1
PSGLSPSLCP.USPT,PGPB.	2
((PSGL$) AND (ATHEROSCLEROSIS OR ARTERIOSCLERORIS)).USPT,PGPB.	24

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Search Results - Record(s) 11 through 20 of 24 returned.

☐ 11. 6492332 . 11 Sep 00; 10 Dec 02. Irrigation solution and methods for inhibition of tumor cell adhesion, pain and inflammation. Demopulos; Gregory A., et al. 514/12; 514/217 514/226.2 514/25 514/254.06 514/259.1 514/263.1 514/266.1 514/280 514/288 514/317 514/327 514/353 514/356 514/397 514/413 514/415 514/509 514/619 514/654 514/680. A61K038/00 A61K031/70 A61K031/55 A61K031/54 A61K031/495 A61K031/505.

☐ 12. 6395882 . 03 Feb 99; 28 May 02. Selectin ligands. Rosen; Steven D., et al. 530/395; 530/350. C07K014/705.

☐ 13. 6380371 . 13 Sep 99; 30 Apr 02. Endoglycan: a novel protein having selectin ligand and chemokine presentation activity. Sassetti; Christopher M., et al. 536/23.1; 530/350 530/380 536/23.5. C07H021/04 C07H001/00.

☐ 14. 6365715 . 01 Nov 99; 02 Apr 02. Human cardiac/brain tolloid-like protein. Arleth; Anthony J, et al. 530/350; 530/399. C07K014/435 C07K014/475.

☐ 15. 6299897 . 15 Nov 99; 09 Oct 01. Inhibition of selectin binding. Nagy; Jon O., et al. 424/443; 424/450 514/23 514/25 514/53 514/54 514/61 514/62 536/1.11 536/17.2 536/18.7 536/4.1 536/53 536/55 536/55.1 536/55.2. A61K009/70 A61K031/715.

☐ 16. 6254852 . 16 Jul 99; 03 Jul 01. Porous inorganic targeted ultrasound contrast agents. Glajch; Joseph L, et al. 424/9.52;. A61B008/00.

☐ 17. 6235309 . 27 Feb 98; 22 May 01. Inhibition of cell-cell binding by lipid assemblies. Nagy; Jon O., et al. 424/450; 514/25 514/42 514/53 514/54 514/61. A61K009/127.

☐ 18. 6124267 . 20 Apr 98; 26 Sep 00. O-glycan inhibitors of selectin mediated inflammation derived from PSGL-1. McEver; Rodger P., et al. 514/25; 514/54 514/62 536/17.2 536/18.7. A61K031/70.

☐ 19. 6123923 . 18 Dec 97; 26 Sep 00. Optoacoustic contrast agents and methods for their use. Unger; Evan C., et al. 424/9.52; 424/450 424/9.1 424/9.2 424/9.3 424/9.6 514/410. A61K049/00 A61K049/22.

☐ 20. 6008017 . 16 Dec 97; 28 Dec 99. Human cardiac/brain tolloid-like protein. Arleth; Anthony J, et al. 435/69.1; 435/320.1 435/325 435/6 536/23.5. C12N015/12.

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Term	Documents
ATHEROSCLEROSIS.USPT,PGPB.	15316
ATHEROSCLEROSES.USPT,PGPB.	30
ARTERIOSCLERORIS.USPT,PGPB.	1
ARTERIOSCLERORI	0
PSGL$	0
PSGL.USPT,PGPB.	84
PSGLB1.USPT,PGPB.	1
"PSGLB1/MIGG.SUB.2B".USPT,PGPB.	1
PSGLITSPNW.USPT,PGPB.	2
PSGLL.USPT,PGPB.	1
PSGLSPSLCP.USPT,PGPB.	2
((PSGL$) AND (ATHEROSCLEROSIS OR ARTERIOSCLERORIS)).USPT,PGPB.	24

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Search Results - Record(s) 21 through 24 of 24 returned.

☐ 21. 5985852 . 16 Feb 99; 16 Nov 99. Inhibition of selectin binding. Nagy; Jon O., et al. 514/54; 424/450 514/23 514/25 514/53 514/61 514/62 536/1.11 536/17.2 536/18.7 536/4.1 536/53 536/55 536/55.1 536/55.2. A61K031/715 A61K009/127 C07H001/00.

☐ 22. 5977080 . 08 Jan 98; 02 Nov 99. Sulfated disaccharide inhibitors of selectins, methods for synthesis and therapeutic use. Rosen; Steven D., et al. 514/25; 514/53 514/61 536/123.13 536/124 536/18.5 536/4.1. A61K031/70 C07H015/00.

☐ 23. 5962422 . 28 Feb 97; 05 Oct 99. Inhibition of selectin binding. Nagy; Jon O., et al. 514/25; 435/7.1 435/7.2 514/42 514/53 514/54 514/61. A61K031/70 G01N033/53.

☐ 24. 5783693 . 23 Aug 95; 21 Jul 98. Methods for synthesizing sulfated disaccharide inhibitors of selectins. Bertozzi; Carolyn, et al. 536/124; 536/123.13 536/18.5 536/4.1. C07H001/00 C07H015/00.

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Term	Documents
ATHEROSCLEROSIS.USPT,PGPB.	15316
ATHEROSCLEROSES.USPT,PGPB.	30
ARTERIOSCLERORIS.USPT,PGPB.	1
ARTERIOSCLERORI	0
PSGL$	0
PSGL.USPT,PGPB.	84
PSGLB1.USPT,PGPB.	1
"PSGLB1/MIGG.SUB.2B".USPT,PGPB.	1
PSGLITSPNW.USPT,PGPB.	2
PSGLL.USPT,PGPB.	1
PSGLSPSLCP.USPT,PGPB.	2
((PSGL$) AND (ATHEROSCLEROSIS OR ARTERIOSCLERORIS)).USPT,PGPB.	24

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Search Results - Record(s) 1 through 10 of 21 returned.

☐ 1. 20020127691 . 27 Nov 01. 12 Sep 02. Highly purified mocarhagin, a cobra venom protease, polynucleotides encoding same and related proteases, and therapeutic uses thereof. Boodhoo, Amechand, et al. 435/226; 435/320.1 435/325 435/69.1 536/23.2 C12N009/64 C07H021/04 C12P021/02 C12N005/06.

☐ 2. 20020061863 . 02 Nov 01. 23 May 02. Novel, specific inhibitors of acute and chronic inflammation. Uppugunduri, Srinivas. 514/49; 514/269 514/738 A61K031/7115 A61K031/513 A61K031/047.

☐ 3. 20020040008 . 18 Jun 01. 04 Apr 02. Method for treating and preventing atherosclerosis. Wagner, Denisa D., et al. 514/41; 424/130.1 514/54 514/8 A61K039/395 A61K031/715 A61K038/16.

☐ 4. 20020037840 . 23 Mar 01. 28 Mar 02. Novel P-selectin glycoprotein ligand (PSGL-1) binding protein and uses therefor. Lorenz, Meike, et al. 514/8; 435/325 435/6 435/69.1 530/395 536/23.5 C12Q001/68 A61K038/16 C07H021/04 C12P021/02 C12N005/06.

☐ 5. 20020031508 . 17 May 01. 14 Mar 02. Methods for diagnosing and treating hemostatic disorders by modulating P-selectin activity. Wagner, Denisa D., et al. 424/94.63; 424/145.1 514/12 A61K038/48 A61K039/395 A61K038/17.

☐ 6. 20020025923 . 18 May 01. 28 Feb 02. Novel selection ligands. Rosen, Steven D., et al. 514/8; 530/395 A61K038/17 C07K014/435.

☐ 7. 20010046970 . 22 Jun 01. 29 Nov 01. Inhibition of selectin binding. Nagy, Jon O., et al. 514/53; 514/54 A61K031/726 A61K031/715.

☐ 8. 20010036931 . 27 Apr 01. 01 Nov 01. Inhibition of cell-cell binding by lipid assemblies. Nagy, Jon O., et al. 514/53; A61K031/7016.

☐ 9. 6492332 . 11 Sep 00; 10 Dec 02. Irrigation solution and methods for inhibition of tumor cell adhesion, pain and inflammation. Demopulos; Gregory A., et al. 514/12; 514/217 514/226.2 514/25 514/254.06 514/259.1 514/263.1 514/266.1 514/280 514/288 514/317 514/327 514/353 514/356 514/397 514/413 514/415 514/509 514/619 514/654 514/680. A61K038/00 A61K031/70 A61K031/55 A61K031/54 A61K031/495 A61K031/505.

☐ 10. 6413936 . 30 Oct 96; 02 Jul 02. Glycomimetics as selectin antagonists and pharmaceuticals having antiinflammatory activity. Schmidt; Wolfgang, et al. 514/23; 514/8 514/9 536/1.11 549/200 549/356 562/459 585/275. A61K031/70.

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Term	Documents
RESTENOSIS.USPT,PGPB.	8198
RESTENOSES.USPT,PGPB.	113
PSGL$	0
PSGL.USPT,PGPB.	84
PSGLB1.USPT,PGPB.	1
"PSGLB1/MIGG.SUB.2B".USPT,PGPB.	1
PSGLITSPNW.USPT,PGPB.	2
PSGLL.USPT,PGPB.	1
PSGLSPSLCP.USPT,PGPB.	2
PSGLU.USPT,PGPB.	1
PSGL1.USPT,PGPB.	5
((PSGL$) AND (RESTENOSIS)).USPT,PGPB.	21

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☐ 11. 6413760 . 18 Feb 98; 02 Jul 02. Highly purified mocarhagin cobra venom protease polynucleotides endcoding same and related proteases and therapeutic uses thereof. Boodhoo; Amechand, et al. 435/226; 435/252.3 435/320.1 435/325 536/23.1 536/23.2. C12N009/64 C12N015/57.

☐ 12. 6395882 . 03 Feb 99; 28 May 02. Selectin ligands. Rosen; Steven D., et al. 530/395; 530/350. C07K014/705.

☐ 13. 6380371 . 13 Sep 99; 30 Apr 02. Endoglycan: a novel protein having selectin ligand and chemokine presentation activity. Sassetti; Christopher M., et al. 536/23.1; 530/350 530/380 536/23.5. C07H021/04 C07H001/00.

☐ 14. 6365715 . 01 Nov 99; 02 Apr 02. Human cardiac/brain tolloid-like protein. Arleth; Anthony J, et al. 530/350; 530/399. C07K014/435 C07K014/475.

☐ 15. 6299897 . 15 Nov 99; 09 Oct 01. Inhibition of selectin binding. Nagy; Jon O., et al. 424/443; 424/450 514/23 514/25 514/53 514/54 514/61 514/62 536/1.11 536/17.2 536/18.7 536/4.1 536/53 536/55 536/55.1 536/55.2. A61K009/70 A61K031/715.

☐ 16. 6254852 . 16 Jul 99; 03 Jul 01. Porous inorganic targeted ultrasound contrast agents. Glajch; Joseph L, et al. 424/9.52;. A61B008/00.

☐ 17. 6235309 . 27 Feb 98; 22 May 01. Inhibition of cell-cell binding by lipid assemblies. Nagy; Jon O., et al. 424/450; 514/25 514/42 514/53 514/54 514/61. A61K009/127.

☐ 18. 6197752 . 05 Sep 96; 06 Mar 01. Glycomimetics as selectin antagonists and pharmaceuticals having antiinflammatory activity prepared therefrom. Schmidt; Wolfgang, et al. 514/23; 536/1.11 536/124 536/18.7. A61K031/70 C07H001/00.

☐ 19. 6008017 . 16 Dec 97; 28 Dec 99. Human cardiac/brain tolloid-like protein. Arleth; Anthony J, et al. 435/69.1; 435/320.1 435/325 435/6 536/23.5. C12N015/12.

☐ 20. 5985852 . 16 Feb 99; 16 Nov 99. Inhibition of selectin binding. Nagy; Jon O., et al. 514/54; 424/450 514/23 514/25 514/53 514/61 514/62 536/1.11 536/17.2 536/18.7 536/4.1 536/53 536/55 536/55.1 536/55.2. A61K031/715 A61K009/127 C07H001/00.

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Term	Documents
RESTENOSIS.USPT,PGPB.	8198
RESTENOSES.USPT,PGPB.	113
PSGL$	0
PSGL.USPT,PGPB.	84
PSGLB1.USPT,PGPB.	1
"PSGLB1/MIGG.SUB.2B".USPT,PGPB.	1
PSGLITSPNW.USPT,PGPB.	2
PSGLL.USPT,PGPB.	1
PSGLSPSLCP.USPT,PGPB.	2
PSGLU.USPT,PGPB.	1
PSGL1.USPT,PGPB.	5
((PSGL$) AND (RESTENOSIS)).USPT,PGPB.	21

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☐ 21. 5962422 . 28 Feb 97; 05 Oct 99. Inhibition of selectin binding. Nagy; Jon O., et al. 514/25; 435/7.1 435/7.2 514/42 514/53 514/54 514/61. A61K031/70 G01N033/53.

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Term	Documents
RESTENOSIS.USPT,PGPB.	8198
RESTENOSES.USPT,PGPB.	113
PSGL$	0
PSGL.USPT,PGPB.	84
PSGLB1.USPT,PGPB.	1
"PSGLB1/MIGG.SUB.2B".USPT,PGPB.	1
PSGLITSPNW.USPT,PGPB.	2
PSGLL.USPT,PGPB.	1
PSGLSPSLCP.USPT,PGPB.	2
PSGLU.USPT,PGPB.	1
PSGL1.USPT,PGPB.	5
((PSGL$) AND (RESTENOSIS)).USPT,PGPB.	21

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P-SELECTIN.USPT,PGPB.	792
P-SELECTINS.USPT,PGPB.	74
PADGEM.USPT,PGPB.	257
PADGEMS	0
GMP-140.USPT,PGPB.	425
GMP-140S	0
GMP140.USPT,PGPB.	119
GMP140S	0
ATHEROSCLEROSIS.USPT,PGPB.	15316
ATHEROSCLEROSES.USPT,PGPB.	30
ARTERIOSCLERORIS.USPT,PGPB.	1
(('P-SELECTIN' OR PADGEM OR 'GMP-140' OR 'GMP140') SAME (ATHEROSCLEROSIS OR ARTERIOSCLERORIS OR RESTENOSIS)).USPT,PGPB.	70

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EPO Abstracts Database
Derwent World Patents Index
IBM Technical Disclosure Bulletins

Search: L6

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Search History

DATE: Friday, May 23, 2003 Printable Copy Create Case

Set Name side by side	Query	Hit Count	Set Name result set
	DB=USPT,PGPB; PLUR=YES; OP=ADJ		
L6	('p-selectin' or padgem or 'gmp-140' or 'gmp140') same (atherosclerosis or arterioscleroris or restenosis)	70	L6
L5	('p-selectin' or padgem or 'gmp-140' or 'gmp140') and (atherosclerosis or arterioscleroris or restenosis)	390	L5
L4	(psgl$) and (atherosclerosis or arterioscleroris)	24	L4
L3	(psgl$) and (restenosis)	21	L3
L2	(psgl$) same (restenosis)	0	L2
L1	wagner-denisa$	4	L1

END OF SEARCH HISTORY

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Search Results -

Term	Documents
"P-SELECTIN GLYCOPROTEIN LIGAND$".USPT,PGPB.	0
PSGL$	0
PSGL.USPT,PGPB.	84
PSGLB1.USPT,PGPB.	1
"PSGLB1/MIGG.SUB.2B".USPT,PGPB.	1
PSGLITSPNW.USPT,PGPB.	2
PSGLL.USPT,PGPB.	1
PSGLSPSLCP.USPT,PGPB.	2
PSGLU.USPT,PGPB.	1
PSGL1.USPT,PGPB.	5
"PSGL1/MIGG.SUB.2B".USPT,PGPB.	2
(L6 AND (PSGL$ OR 'P-SELECTIN GLYCOPROTEIN LIGAND$')).USPT,PGPB.	15

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US Pre-Grant Publication Full-Text Database
JPO Abstracts Database
EPO Abstracts Database
Derwent World Patents Index
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Search: L7

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Search History

DATE: Friday, May 23, 2003 Printable Copy Create Case

Set Name side by side	Query	Hit Count	Set Name result set
	DB=USPT,PGPB; PLUR=YES; OP=ADJ		
L7	L6 and (psgl$ or 'p-selectin glycoprotein ligand$')	15	L7
L6	('p-selectin' or padgem or 'gmp-140' or 'gmp140') same (atherosclerosis or arterioscleroris or restenosis)	70	L6
L5	('p-selectin' or padgem or 'gmp-140' or 'gmp140') and (atherosclerosis or arterioscleroris or restenosis)	390	L5
L4	(psgl$) and (atherosclerosis or arterioscleroris)	24	L4
L3	(psgl$) and (restenosis)	21	L3
L2	(psgl$) same (restenosis)	0	L2
L1	wagner-denisa$	4	L1

END OF SEARCH HISTORY

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Search Results - Record(s) 1 through 10 of 15 returned.

☐ 1. 20030082143 . 12 Jun 02. 01 May 03. Receptor-mediated gene delivery using bacteriophage vectors. Larocca, David, et al. 424/93.2; 435/456 514/44 A61K048/00 C12N015/86.

☐ 2. 20030040496 . 17 May 01. 27 Feb 03. Methods for halting unwanted cell growth, such as using ligand-directed nucleic acid delivery vehicles. Chandler, Lois Ann, et al. 514/44; 424/143.1 514/8 A61K048/00 A61K039/395.

☐ 3. 20020168338 . 23 Oct 98. 14 Nov 02. COMPOSITIONS AND METHODS FOR DELIVERY OF AGENTS FOR NEURONAL REGENERATION AND SURVIVAL. BAIRD, ANDREW. 424/93.2; 424/193.1 424/423 424/424 424/425 424/468 424/469 424/486 435/320.1 514/44 536/24.1 536/24.5 A61K048/00 C07H021/04 A61K039/385.

☐ 4. 20020061863 . 02 Nov 01. 23 May 02. Novel, specific inhibitors of acute and chronic inflammation. Uppugunduri, Srinivas. 514/49; 514/269 514/738 A61K031/7115 A61K031/513 A61K031/047.

☐ 5. 20020058034 . 12 Jul 01. 16 May 02. Inhibition of differentiation of cytotoxic T-cells by P-selectin ligand (PSGL) antagonists. Manjunath, Narasimhaswamy, et al. 424/144.1; 514/12 514/54 A61K039/395 A61K038/17 A61K031/726.

☐ 6. 20020040008 . 18 Jun 01. 04 Apr 02. Method for treating and preventing atherosclerosis. Wagner, Denisa D., et al. 514/41; 424/130.1 514/54 514/8 A61K039/395 A61K031/715 A61K038/16.

☐ 7. 20020037840 . 23 Mar 01. 28 Mar 02. Novel P-selectin glycoprotein ligand (PSGL-1) binding protein and uses therefor. Lorenz, Meike, et al. 514/8; 435/325 435/6 435/69.1 530/395 536/23.5 C12Q001/68 A61K038/16 C07H021/04 C12P021/02 C12N005/06.

☐ 8. 20020031508 . 17 May 01. 14 Mar 02. Methods for diagnosing and treating hemostatic disorders by modulating P-selectin activity. Wagner, Denisa D., et al. 424/94.63; 424/145.1 514/12 A61K038/48 A61K039/395 A61K038/17.

☐ 9. 6551618 . 23 Oct 98; 22 Apr 03. Compositions and methods for delivery of agents for neuronal regeneration and survival. Baird; Andrew, et al. 424/484; 424/468 424/469 424/486 435/320.1 435/455 435/91.4 514/44. A61K009/14 A61K048/00.

☐ 10. 6492332 . 11 Sep 00; 10 Dec 02. Irrigation solution and methods for inhibition of tumor cell adhesion, pain and inflammation. Demopulos; Gregory A., et al. 514/12; 514/217 514/226.2 514/25 514/254.06 514/259.1 514/263.1 514/266.1 514/280 514/288 514/317 514/327 514/353 514/356 514/397 514/413 514/415 514/509 514/619 514/654 514/680. A61K038/00 A61K031/70 A61K031/55 A61K031/54 A61K031/495 A61K031/505.

Generate Collection | Print

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"PSGLB1/MIGG.SUB.2B".USPT,PGPB.	1
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PSGLL.USPT,PGPB.	1
PSGLSPSLCP.USPT,PGPB.	2
PSGLU.USPT,PGPB.	1
PSGL1.USPT,PGPB.	5
"PSGL1/MIGG.SUB.2B".USPT,PGPB.	2
(L6 AND (PSGL$ OR 'P-SELECTIN GLYCOPROTEIN LIGAND$')).USPT,PGPB.	15

There are more results than shown above. Click here to view the entire set.

Previous Page Next Page

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Search Results - Record(s) 11 through 15 of 15 returned.

☐ 11. 6448083 . 26 Feb 99; 10 Sep 02. Receptor-mediated gene delivery using bacteriophage vectors. Larocca; David, et al. 435/456; 435/320.1. C12N015/64 C12N015/63.

☐ 12. 6380371 . 13 Sep 99; 30 Apr 02. Endoglycan: a novel protein having selectin ligand and chemokine presentation activity. Sassetti; Christopher M., et al. 536/23.1; 530/350 530/380 536/23.5. C07H021/04 C07H001/00.

☐ 13. 6251599 . 06 Nov 98; 26 Jun 01. Stabilized nucleic acid compositions and methods of preparation and use thereof. Chen; Xian, et al. 435/6; 514/44 536/23.1 536/23.4 536/23.5 536/24.5. C12Q001/68.

☐ 14. 6124267 . 20 Apr 98; 26 Sep 00. O-glycan inhibitors of selectin mediated inflammation derived from PSGL-1. McEver; Rodger P., et al. 514/25; 514/54 514/62 536/17.2 536/18.7. A61K031/70.

☐ 15. 6054312 . 29 Aug 97; 25 Apr 00. Receptor-mediated gene delivery using bacteriophage vectors. Larocca; David, et al. 435/320.1; 530/350 530/387.1 536/23.5 536/23.72. C12N015/63 C12N015/33 C12N015/12 C07K014/00.

Generate Collection | Print

Term	Documents
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(L6 AND (PSGL$ OR 'P-SELECTIN GLYCOPROTEIN LIGAND$')).USPT,PGPB.	15

There are more results than shown above. Click here to view the entire set.

Previous Page Next Page

WEST

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Main Menu | Search Form | Posting Counts | Show S Numbers | Edit S Numbers | Preferences | Cases

Search Results -

Term	Documents
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There are more results than shown above. Click here to view the entire set.

Database: US Patents Full-Text Database
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JPO Abstracts Database
EPO Abstracts Database
Derwent World Patents Index
IBM Technical Disclosure Bulletins

Search: L7

Refine Search

Recall Text | Clear

Search History

DATE: Friday, May 23, 2003 Printable Copy Create Case

Set Name side by side	Query	Hit Count	Set Name result set
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L7	L6 and (psgl$ or 'p-selectin glycoprotein ligand$')	15	L7
L6	('p-selectin' or padgem or 'gmp-140' or 'gmp140') same (atherosclerosis or arterioscleroris or restenosis)	70	L6
L5	('p-selectin' or padgem or 'gmp-140' or 'gmp140') and (atherosclerosis or arterioscleroris or restenosis)	390	L5
L4	(psgl$) and (atherosclerosis or arterioscleroris)	24	L4
L3	(psgl$) and (restenosis)	21	L3
L2	(psgl$) same (restenosis)	0	L2
L1	wagner-denisa$	4	L1

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               9  S4
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...completed examining records
      S6       2  RD S5 (unique items)
? t s6/3/all


 6/3/1     (Item 1 from file: 5)
DIALOG(R)File   5:Biosis Previews(R)
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13115159   BIOSIS NO.: 200100322308
Alterations in platelet thrombus formation, leukocyte recruitment, and
  intimal hyperplasia in P-selectin-deficient mice after transluminal
  femoral artery injury.
AUTHOR: Smyth Susan S(a); Reis Ernane D; Fallon John T; Gordon Ron; Coller
  Barry S(a)
AUTHOR ADDRESS: (a)Medicine, Mount Sinai School of Medicine, New York, NY**
  USA
JOURNAL: Blood  96 (11 Part 1):p813a November 16, 2000
MEDIUM: print
CONFERENCE/MEETING: 42nd Annual Meeting of the American Society of
Hematology  San Francisco, California, USA  December 01-05, 2000
SPONSOR: American Society of Hematology
ISSN: 0006-4971
RECORD TYPE: Abstract
LANGUAGE: English
SUMMARY LANGUAGE: English


 6/3/2     (Item 1 from file: 399)
DIALOG(R)File 399:CA SEARCH(R)
(c) 2001 AMERICAN CHEMICAL SOCIETY. All rts. reserv.

  124135703    CA: 124(11)135703f    PATENT
  Method using agents inhibiting interaction between P-selectin??? and a
P-selectin ligand for treating and preventing atherosclerosis
  INVENTOR(AUTHOR): Wagner, Denisa D.; Johnson, Robert C.
  LOCATION: USA
  ASSIGNEE: Center for Blood Research, Inc.
  PATENT: PCT International ; WO 9533484 A1  DATE: 951214
  APPLICATION: WO 95US6940 (950601) *US 253663 (940603) *US 377798 (950124)
  PAGES: 35 pp.  CODEN: PIXXD2  LANGUAGE: English  CLASS: A61K-039/395A;
A61K-038/02B; A61K-038/16B; A61K-031/70B  DESIGNATED COUNTRIES: CA; JP
  DESIGNATED REGIONAL: AT; BE; CH; DE; DK; ES; FR; GB; GR; IE; IT; LU; MC;
NL; PT; SE
? s restenosis and atherosclerosis

          24623  RESTENOSIS
         136871  ATHEROSCLEROSIS
      S7    3360  RESTENOSIS AND ATHEROSCLEROSIS
? s s7 and review?

           3360  S7
        3019137  REVIEW?
      S8     495  S7 AND REVIEW?
? s restenosis (20n) atherosclerosis and review?

          24623  RESTENOSIS
         136871  ATHEROSCLEROSIS
           1703  RESTENOSIS(20N)ATHEROSCLEROSIS
        3019137  REVIEW?
      S9     303  RESTENOSIS (20N) ATHEROSCLEROSIS AND REVIEW?
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restenosis and atherosclerosis

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          24623  RESTENOSIS
         136871  ATHEROSCLEROSIS
      S7    3360  RESTENOSIS AND ATHEROSCLEROSIS
? s s7 and review?

          3360  S7
       3019137  REVIEW?
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? s restenosis (20n) atherosclerosis and review?

          24623  RESTENOSIS
         136871  ATHEROSCLEROSIS
           1703  RESTENOSIS(20N)ATHEROSCLEROSIS
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      S9    303  RESTENOSIS (20N) ATHEROSCLEROSIS AND REVIEW?
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            303  S9
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 11/3/1     (Item 1 from file: 73)
DIALOG(R)File  73:EMBASE
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10863398     EMBASE No: 2000345938
  Roles of P-selectin in inflammation, neointimal formation,
and vascular remodeling in balloon-injured rat carotid arteries
  Hayashi S.-I.; Watanabe N.; Nakazawa K.; Suzuki J.; Tsushima K.; Tamatani
T.; Sakamoto S.; Isobe M.
  Dr. M. Isobe, Dept. of Cardiovascular Medicine, Tokyo Medical and Dental
  University, 1-5-45 Yushima, Bunkyo-ku, Tokyo 113-8519  Japan
  AUTHOR EMAIL: isobemi.med3@med.tmd.ac.jp
  Circulation ( CIRCULATION ) (United States)  03 OCT 2000, 102/14
  (1710-1717)
  CODEN: CIRCA   ISSN: 0009-7322
  DOCUMENT TYPE: Journal; Review
  LANGUAGE: ENGLISH   SUMMARY LANGUAGE: ENGLISH
  NUMBER OF REFERENCES: 24
? t s11/7/all


 11/7/1     (Item 1 from file: 73)
DIALOG(R)File  73:EMBASE
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10863398 EMBASE No: 2000345938
Roles of **P-selectin** in inflammation, neointimal formation, and vascular remodeling in balloon-injured rat carotid arteries
Hayashi S.-I.; Watanabe N.; Nakazawa K.; Suzuki J.; Tsushima K.; Tamatani T.; Sakamoto S.; Isobe M.
Dr. M. Isobe, Dept. of Cardiovascular Medicine, Tokyo Medical and Dental University, 1-5-45 Yushima, Bunkyo-ku, Tokyo 113-8519 Japan
AUTHOR EMAIL: isobemi.med3@med.tmd.ac.jp
Circulation (CIRCULATION) (United States) 03 OCT 2000, 102/14 (1710-1717)
CODEN: CIRCA ISSN: 0009-7322
DOCUMENT TYPE: Journal; Review
LANGUAGE: ENGLISH SUMMARY LANGUAGE: ENGLISH
NUMBER OF REFERENCES: 24

Background - **P-selectin** plays key roles in mediating inflammation through promoting adherence of leukocytes to activated platelets and endothelium. This process is one of the initial events in **atherosclerosis** and **restenosis** after coronary angioplasty.
Methods and Results - Using a rat balloon-injury model, we examined the role of **P-selectin** in vascular inflammatory processes. In the acute phase, immunohistochemistry revealed that **P-selectin** was intensely expressed on both activated platelets covering the denuded segment and endothelial cells of the inflamed adventitial small vessels. Treatment with an anti-**P-selectin** monoclonal antibody (MAb) for 8 consecutive days significantly inhibited neointimal formation at day 14 (42% inhibition; $P<0.05$), and this effect persisted at day 56 (40% inhibition; $P<0.01$) compared with the control group. Vascular shrinking accompanying adventitial fibrosis was also attenuated at day 56. Inhibition of both neointimal formation and vascular shrinking resulted in the lumen area of the anti-**P-selectin** treatment group being approx. eq.3 times larger at day 56 than that of the control group. Accumulation of CD45-positive leukocytes in the developing neointima, media, and adventitia at day 8 was significantly inhibited by treatment with the anti-**P-selectin** MAb. Scanning electron microscopy demonstrated that anti-**P-selectin** treatment resulted in a less thrombogenic surface of the arterial intima, which featured a pseudoendothelial appearance at day 14 after injury. Conclusions - These results suggest that inhibition of **P-selectin**-mediated leukocyte recruitment prevents the development of neointimal formation, adventitial inflammation, and vascular shrinking and promotes pseudoendothelialization by luminal smooth muscle cells. This treatment thus beneficially affects vascular remodeling after balloon injury in rats.
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Set	Items	Description
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S5	2	S2 AND S4
S6	2	RD S5 (unique items)
S7	3360	RESTENOSIS AND ATHEROSCLEROSIS
S8	495	S7 AND REVIEW?
S9	303	RESTENOSIS (20N) ATHEROSCLEROSIS AND REVIEW?
S10	0	S9 AND P(W)SELECTION?
S11	1	S9 AND P(W)SELECTIN?

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  File 399:CA SEARCH(R)  1967-2001/UD=13520
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Processing
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                  (ATHEROSCLER?)
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...completed examining records
      S2      14  RD S1 (unique items)
? t s2/7/all


 2/7/1     (Item 1 from file: 5)
DIALOG(R)File   5:Biosis Previews(R)
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13115159 BIOSIS NO.: 200100322308

Alterations in platelet thrombus formation, leukocyte recruitment, and intimal hyperplasia in P-selectin-deficient mice after transluminal femoral artery injury.

AUTHOR: Smyth Susan S(a); Reis Ernane D; Fallon John T; Gordon Ron; Coller Barry S(a)

AUTHOR ADDRESS: (a)Medicine, Mount Sinai School of Medicine, New York, NY** USA

JOURNAL: Blood 96 (11 Part 1):p813a November 16, 2000

MEDIUM: print

CONFERENCE/MEETING: 42nd Annual Meeting of the American Society of Hematology San Francisco, California, USA December 01-05, 2000

SPONSOR: American Society of Hematology

ISSN: 0006-4971

RECORD TYPE: Abstract

LANGUAGE: English

SUMMARY LANGUAGE: English

ABSTRACT: Platelet activation at the site of vascular injury, such as occurs after **atherosclerotic** plaque rupture or following percutaneous intervention, results in platelet-neutrophil interactions, which contribute to local thrombosis, downstream microcirculatory events, and systemic inflammation. The initial interaction of activated platelets with neutrophils is mediated by platelet P-selectin binding to neutrophil **PSGL**-1. To investigate the role of platelet-neutrophil interactions in response to arterial injury, we performed transluminal, wire-induced injury to the femoral artery of wild-type C57B1/6 mice (n=26) and P-selectin -/- mice (n=26). The femoral arteries of anesthesized male mice aged 8-10 weeks were injured by passing a 0.25 mm wire in the lumen of the femoral artery three times, and the mice were euthanized by perfusion fixation 1 h or 4 weeks after injury. 1 h after injury in wild-type mice, platelets were found adherent to the blood vessel wall and neutrophils were attached to the platelets. As viewed by TEM and SEM, the platelet layer varied between 1 and apprxeq3 platelets thick, and many of the platelets in contact with the wall were spread and at least partially degranulated. 1 h after injury of P-selectin -/- mice, the platelet layer appeared less compact and appeared to extend further into the lumen; moreover, more of the platelets appeared to retain their granules. There was a striking decrease in leukocyte attachment to the platelets. Four weeks after injury, the neointimal area in P-selectin -/- mice (2,100 +- 900 mum2) was significantly smaller than in the wild-type mice (10,200 +- 2,100 mum2) (p=0.004). These results indicate that P-selectin is required for neutrophil recruitment to platelets lining the

vessel wall 1 h after injury and P-selectin deficiency protects mice from developing intimal hyperplasia 4 weeks after injury. Moreover, in the P-selectin -/- mice, the platelets depositing on the damaged vessel appeared to be less activated and to extend further into the lumen, suggesting that P-selectin may play a role in platelet activation and platelet thrombus formation. Our data support the possibility that antagonists to P-selectin may decrease intimal hyperplasia and clinical restenosis after percutaneous vascular interventions in humans.

2/7/2 (Item 2 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

13092476 BIOSIS NO.: 200100299625
Chemokines immobilized on early **atherosclerotic** endothelium mediate monocyte arrest via VLA-4.
AUTHOR: Huo Yuqing(a); Weber Christian; Ley Klaus(a)
AUTHOR ADDRESS: (a)University of Virginia, Health Science Center, Charlottesville, VA, 22908**USA
JOURNAL: FASEB Journal 15 (4):pA584 March 7, 2001
MEDIUM: print
CONFERENCE/MEETING: Annual Meeting of the Federation of American Societies for Experimental Biology on Experimental Biology 2001 Orlando, Florida, USA March 31-April 04, 2001
ISSN: 0892-6638
RECORD TYPE: Abstract
LANGUAGE: English
SUMMARY LANGUAGE: English

ABSTRACT: In reconstituted models using cultured endothelial cells or plate surfaces coated with chemokines and adhesion molecules, leukocyte arrest can be triggered by chemokines such as IL-8, MIP-2, GRO-alpha, RANTES and SDF-1alpha. Here, we investigate which chemokines immobilized on the spontaneous early **atherosclerotic** lesions can contribute to the arrest of rolling monocytes in **atherosclerosis**. This study was performed on a novel ex vivo model, in which monocyte roll and arrest on lesion-prone sites in carotid arteries from apoE-/-, but not control mice mainly through P-selectin/**PSGL**-1 and VLA-4/VCAM-1 (Huo et al., Circ. Res. 87: 146-152, 2000). Monocyte arrest in this model is blocked by 55% by pertussis toxin, by 37% with the CCR-1, 3 and 5 inhibitor, metRANTES, and by 42% with the CXCR-2 inhibitor, 8-73 GRO-alpha, but not by the CCR-2 inhibitor, 9-79 MCP-1. RANTES, KC (mouse GRO-alpha) and MCP-1 are expressed on the endothelium of arteries from apoE-/-, but not wild-type mice, and antibody to KC block arrest by 42%. Perfusing KC or RANTES through carotid arteries with early **atherosclerotic** lesions can further increased the number of arresting monocyte, while perfusing MCP-1 had no effect. Blockade of VLA-4/VCAM-1 but not CD18/ICAM-1 dramatically inhibited the arrest of monocyte both under physiological and KC or RANTES perfused conditions. These results suggest that KC and RANTES immobilized on early **atherosclerotic** lesions activate VLA-4 on monocytes to mediate efficient monocyte arrest on the luminal surface of lesion prone arteries of apoE-/- mice. Surprisingly, MCP-1 does not function as an arrest chemokines under physiologic conditions. Our findings clarify the molecular mechanism involved in monocyte recruitment to **atherosclerotic** lesions and suggest potential new approaches to curbing the development of **atherosclerosis** lesions.

2/7/3 (Item 3 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

12864535 BIOSIS NO.: 200100071684
Complex roles of P-selectin and von Willebrand factor in inflammation and hemostasis.

AUTHOR: Wagner D D(a); Andre P(a); Denis C V(a); Hartwell D W(a);
Hrachovinova I(a); Methia N(a)
AUTHOR ADDRESS: (a)The Center for Blood Research, Department of Pathology,
Harvard Medical School, Boston, MA**USA
JOURNAL: Journal of Submicroscopic Cytology and Pathology 32 (3):p333
July, 2000
MEDIUM: print
CONFERENCE/MEETING: XIth International Vascular Biology Meeting Geneva,
Switzerland September 05-09, 2000
ISSN: 1122-9497
RECORD TYPE: Citation
LANGUAGE: English
SUMMARY LANGUAGE: English

2/7/4 (Item 4 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

12058582 BIOSIS NO.: 199900339101
Direct demonstration of P-selectin- and VCAM-1-dependent mononuclear cell
rolling in early **atherosclerotic** lesions of apolipoprotein
E-deficient mice.
AUTHOR: Ramos Carroll L; Huo Yuqing; Jung Unsu; Ghosh Shukti; Manka David R
; Sarembock Ian J; Ley Klaus(a)
AUTHOR ADDRESS: (a)Department of Biomedical Engineering, Health Sciences
Center, University of Virginia, Charlottes**USA
JOURNAL: Circulation Research 84 (11):p1237-1244 June 11, 1999
ISSN: 0009-7330
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English
SUMMARY LANGUAGE: English

ABSTRACT: Apolipoprotein E-deficient (ApoE-/-) mice develop
atherosclerotic lesions throughout the arterial tree, including the
carotid bifurcation. Although the expression of adhesion molecules such
as ICAM-1, vascular cell adhesion molecule-1 (VCAM-1), and P-selectin on
endothelium that overlie **atherosclerotic** plaques has been
implicated in monocyte recruitment to developing lesions, monocyte
adhesion in **atherosclerotic** vessels has not been observed directly.
To investigate which adhesion molecules may be important in monocyte
adhesion to **atherosclerotic** lesions, an isolated mouse carotid
artery preparation was developed and perfused with mononuclear cells. We
show rolling and attachment of the human monocytic cell line U937 and the
mouse monocyte-macrophage cell line P388D1 in carotid arteries from 10-
to 12-week-old ApoE-/- and C57BL/6 wild-type mice fed a Western-type diet
(21% fat wt/wt) for 4 to 5 weeks. No rolling was observed in carotid
arteries from C57BL/6 or BALB/c wild-type mice fed a chow diet and little
was observed in BALB/c mice fed a Western-type diet. This model
represents early lesion development as shown by minimal macrophage
infiltration in the intima of carotid arteries from ApoE-/- mice fed a
Western-type diet. Rolling was observed at shear stresses that were
characteristic of the low-shear recirculation zone near the carotid
bifurcation. Mononuclear cell attachment and rolling were significantly
inhibited by monoclonal antibody blockade of P-selectin or its leukocyte
ligand **P-selectin glycoprotein ligand**-1. Rolling
velocities increased after monoclonal antibody blockade of mononuclear
cell alpha4-integrin or VCAM-1, which indicates that alpha4-integrin
interacting with VCAM-1 stabilizes rolling interactions and prolongs
monocyte transit times.

2/7/5 (Item 5 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

11715101 BIOSIS NO.: 199800496832
P-selectin and MAC-1 mediate monocyte rolling and adhesion to ECM-bound platelets under flow conditions.
AUTHOR: Kuijper P H M(a); Tores H I Gallardo; Houben L A M J; Lammers J-W J ; Zwaginga J J; Koenderman L
AUTHOR ADDRESS: (a)Dep. Pulmonary Dis., Room G.03.550, Univ. Hosp. Utrecht, Heidelberglaan 100, 3584 CZ, Utrecht**Netherlands
JOURNAL: Journal of Leukocyte Biology 64 (4):p467-473 Oct., 1998
ISSN: 0741-5400
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English

ABSTRACT: Accumulation of monocyte-derived foam cells in focal areas of the **atherosclerotic** (A.S.-) lesion is one of the key events in early atherogenesis. Using a flow model for the damaged vessel wall, we examined the ability of ECM-bound platelets to induce monocyte tethering and adhesion. Whereas, ECM-proteins alone induced monocyte adhesion only at low shear stresses (< 100 mPa), ECM-bound platelets induced monocyte rolling and adhesion at shear stresses up to 240 mPa. Studies with specific antibodies showed that monocyte adhesion to platelets was mainly mediated by P-selectin and monocyte **PSGL**-1 (maximum inhibition 90%). beta2-Integrin blocking CD18 and CD11b antibodies partly inhibited the arrest of rolling cells. Antibodies against other adhesion molecules such as LFA-1, PECAM-1, and beta1-integrins had no effect. Even sparsely adhered platelets (apprx 10% coverage of the surface) already strongly supported monocyte tethering. In conclusion, activated platelets present on ECM are a powerful adhesive substrate for monocyte recruitment under flow conditions.

2/7/6 (Item 6 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

11486174 BIOSIS NO.: 199800267506
Adhesion of monocytes to vascular cell adhesion molecule-1-transduced human endothelial cells. Implications for atherogenesis.
AUTHOR: Gerszten Robert E; Lim Yaw-Chyn; Ding Han T; Snapp Karen; Kansas Goeffrey; Dichek David A; Cabanas Carlos; Sanchez-Madrid Francisco; Gimbrone Michael A Jr; Rosenzweig Anthony; Luscinskas Francis W(a)
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ABSTRACT: To study the role of vascular cell adhesion molecule-1 (VCAM-1) in monocyte recruitment and atherogenesis, we constructed a recombinant adenovirus, AdRSVrVCAM-1, carrying the rabbit VCAM-1 cDNA. We have previously shown that AdRSVrVCAM-1-transduced human umbilical vein endothelial cells (HUVECs) support the adhesion of CD4+ CD45RO+ memory T lymphocytes under laminar flow conditions. We now demonstrate that AdRSVrVCAM1-transduced HUVECs support the adhesion of peripheral blood monocytes at a shear stress of ltoreq 1.5 dyne/cm2. Although VCAM-1 supported only firm adhesion of lymphocytes, it was able to mediate monocyte rolling, firm adhesion, and transmigration when expressed in the context of otherwise unactivated vascular endothelium. VCAM1-transduced HUVECs supported the adhesion of as many as 4-fold more monocytes than T cells under laminar flow. The greater monocyte adhesion was explained at least in part by leukocyte-leukocyte interactions (secondary adhesions), which were not seen with T cells. These secondary monocyte interactions were specifically blocked by monoclonal antibodies to L-selectin and

P-selectin glycoprotein ligand-1. These data demonstrate that VCAM-1 expressed in the context of unactivated vascular endothelium supports the adhesion of the leukocyte populations present in **atherosclerotic** plaque and may contribute to the predominance of monocytes over lymphocytes.

2/7/7 (Item 7 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

09156353 BIOSIS NO.: 199497164723
Structure/function studies of **P-selectin glycoprotein ligand**.
AUTHOR: Barone Karen M; Pittman Deborah; Shaw Gray
AUTHOR ADDRESS: Genetics Inst. Inc., 87 Cambridge Park Drive, Cambridge, MA 02140**USA
JOURNAL: Journal of Cellular Biochemistry Supplement 0 (18 PART A):p290 1994
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ISSN: 0733-1959
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LANGUAGE: English

2/7/8 (Item 1 from file: 73)
DIALOG(R)File 73:EMBASE

11344635 EMBASE No: 2001358926
Adhesion molecules and atherogenesis
Huo Y.; Ley K.
K. Ley, Department of Biomedical Engineering, University of Virginia, Health Science Center, Charlottesville, VA 22908 United States
Acta Physiologica Scandinavica (ACTA PHYSIOL. SCAND.) (United Kingdom) 2001, 173/1 (35-43)
CODEN: APSCA ISSN: 0001-6772
DOCUMENT TYPE: Journal ; Review
LANGUAGE: ENGLISH SUMMARY LANGUAGE: ENGLISH
NUMBER OF REFERENCES: 56

Atherosclerosis is an inflammatory disease of the vessel wall characterized by monocyte infiltration in response to pro-atherogenic factors such as oxidized lipids. Recently, the role of specific adhesion molecules in this process has been explored. The endothelium overlying **atherosclerotic** lesions expresses P-selectin and the shoulder regions express vascular cell adhesion molecule-1 (VCAM-1) and intercellular adhesion molecule-1 (ICAM-1), which is also expressed on endothelium in regions not prone to plaque development. Serum levels of soluble P-selectin, ICAM-1 and VCAM-1 are elevated in patients with angina pectoris or peripheral **atherosclerotic** disease. Reconstituted in vitro systems using monocytes on cytokine-activated endothelial cells under shear flow suggested the involvement of P-selectin, L-selectin, VCAM-1, its ligand, VLA-4 integrin and CD18 integrins. Studies of monocyte adhesion in isolated perfused carotid arteries harvested from **atherosclerotic** (apoE-/-) mice show a predominant involvement of P-selectin and its ligand P-selectin glycoprotein-1 (**PSGL**-1) in rolling and of VLA-4 and VCAM-1 in firm adhesion. Consistent with these findings, apoE-/- mice that are also deficient for P-selectin show significantly reduced **atherosclerotic** lesion sizes and are almost completely protected from neointimal growth after vascular injury. Milder effects are also seen in the low-density lipoprotein (LDL) receptor deficient (LDLR-/-) mouse. In a high cholesterol/cholate model, a role of ICAM-1 and CD18 integrins was also shown, but this awaits confirmation in more physiologic models. Transient blockade of the VLA-4/VCAM-1 adhesion pathway by antibodies or peptides in

apoE-/- or LDLR-/- mice reduced monocyte and lipid accumulation in lesions. These data suggest that P-selectin, **PSGL**-1, VLA-4 and VCAM-1 are the most important adhesion molecules involved in monocyte recruitment to **atherosclerotic** lesions.

2/7/9 (Item 2 from file: 73)
DIALOG(R)File 73:EMBASE
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11332032 EMBASE No: 2001346318
High-shear-stress-induced activation of platelets and microparticles enhances expression of cell adhesion molecules in THP-1 and endothelial cells
Nomura S.; Tandon N.N.; Nakamura T.; Cone J.; Fukuhara S.; Kambayashi J.
S. Nomura, Otsuka America Pharmaceutical Inc., Rockville, MD 20850 United States
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Atherosclerosis (ATHEROSCLEROSIS) (Ireland) 2001, 158/2 (277-287)
CODEN: ATHSB ISSN: 0021-9150
PUBLISHER ITEM IDENTIFIER: S0021915001004336
DOCUMENT TYPE: Journal ; Article
LANGUAGE: ENGLISH SUMMARY LANGUAGE: ENGLISH
NUMBER OF REFERENCES: 68

Interaction between leukocyte and endothelial cells (ECs) is essential for vascular homeostasis and competent immune-inflammatory responses in vivo. Platelet-derived microparticles (PMPs) are generated by high shear stress and may appear in diseased small arteries and arterioles in various clinical settings. In this study, we used flow cytometry and confocal laser scanning microscopy to investigate the effects of high-shear-induced platelet and microparticle activation in adhesion molecules of THP-1 and ECs. We also measured the production of some cytokines and studied cytokine mRNA from THP-1 and ECs after PMP stimulation. PMP stimulation of THP-1 cells increased CD11b, CD32, and CD33 but not CD29, CD31, and CD36. PMP stimulation of ECs increased CD54 and CD63 but not CD9, CD29, and CD31. PMPs induced interleukin-8 (IL-8), interleukin-1beta (IL-1beta), and tumor necrosis factor alpha (TNFalpha) production by THP-1. PMPs also induced IL-8, IL-1beta, and interleukin-6 (IL-6) production by ECs. Production was time-dependent. With RT-PCR, some cytokine mRNAs were detected in THP-1 and ECs after PMP stimulation. In relation to adhesiveness after PMP stimulation, we could clearly observe a shift in distribution not only of CD11b in THP-1 cells but also of CD54 in ECs. In addition, anti-**P-selectin glycoprotein ligand**-1 antibody reduced the expression of CD11b, CD32, and CD33 in THP-1 after PMP stimulation. These results suggest that high-shear-induced microparticles may contribute to the development of **atherosclerosis** and participate in vascular damage in inflammatory disorders. (c) 2001 Elsevier Science Ireland Ltd.

2/7/10 (Item 3 from file: 73)
DIALOG(R)File 73:EMBASE
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11302989 EMBASE No: 2001317223
Adhesive interactions of leukocytes, platelets, and the vessel wall during hemostasis and inflammation
McEver R.P.
Dr. R.P. McEver, Warren Medical Research Institute, University of Oklahoma, Health Sciences Center, 825 N. E. 13th Street, Oklahoma City, OK 73104 United States
AUTHOR EMAIL: rodger-mcever@ouhsc.edu
Thrombosis and Haemostasis (THROMB. HAEMOST.) (Germany) 2001, 86/3 (746-756)
CODEN: THHAD ISSN: 0340-6245
DOCUMENT TYPE: Journal ; Review

LANGUAGE: ENGLISH
NUMBER OF REFERENCES: 149

2/7/11 (Item 4 from file: 73)
DIALOG(R)File 73:EMBASE

07741206 EMBASE No: 1999223340
P-selectin binding promotes the adhesion of monocytes to VCAM-1 under flow conditions
Yago T.; Tsukuda M.; Minami M.
Dr. M. Minami, Department of Otolaryngology, Yokohama City University, School of Medicine, Fukuura 3-9, Kanazawa-ku, Yokohama 236 Japan
Journal of Immunology (J. IMMUNOL.) (United States) 01 JUL 1999, 163/1 (367-373)
CODEN: JOIMA ISSN: 0022-1767
DOCUMENT TYPE: Journal; Article
LANGUAGE: ENGLISH SUMMARY LANGUAGE: ENGLISH
NUMBER OF REFERENCES: 39

This study examined the adhesive interaction of peripheral blood monocytes with VCAM-1 and analyzed the effect of P-selectin binding to monocytes on the adhesive interaction with VCAM-1 under flow conditions. **P- selectin glycoprotein ligand**-1 is expressed on most monocytes. Furthermore, most monocytes bind soluble P-selectin derived from platelets. P-selectin binding to monocytes did not alter the amount of expression of alphainf 4 integrin on monocytes. However, the mean channel fluorescence value for binding Cy2- conjugated soluble VCAM-1 to P-selectin-bound monocytes was slightly more than that for binding Cy2-conjugated soluble VCAM-1 to untreated monocytes. Under flow conditions, the number of P-selectin-bound monocytes bound to VCAM-1 was much higher than that of untreated monocytes bound to VCAM-1. These bindings were abolished by pretreatment of untreated monocytes and P-selectin-bound monocytes with anti-VCAM-1 mAb or anti-alphainf 4 integrin mAb. Furthermore, P-selectin binding to monocytes increased shear resistance and thus increased the adhesive strength of monocytes to VCAM-1. These findings indicate that P-selectin binding to monocytes enhances the adhesive interaction of monocytes with VCAM-1. It is suggested that **P -selectin glycoprotein ligand**-1/P-selectin interaction and alphainf 4 integrin/VCAM-1 interaction can act sequentially in the adhesion cascade that regulates monocyte trafficking to inflammatory and **atherosclerotic** lesion.

2/7/12 (Item 5 from file: 73)
DIALOG(R)File 73:EMBASE

07381280 EMBASE No: 1998291193
Important contributions of **P-selectin glycoprotein ligand**-1-mediated secondary capture to human monocyte adhesion to P-selectin, E-selectin, and TNF-alpha-activated endothelium under flow in vitro
Lim Y.-C.; Snapp K.; Kansas G.S.; Camphausen R.; Ding H.; Luscinskas F.W.
Dr. F.W. Luscinskas, Brigham and Women's Hospital, 221 Longwood Avenue, Boston, MA 02115 United States
AUTHOR EMAIL: fluscinskas@rics.bwh.harvard.edu
Journal of Immunology (J. IMMUNOL.) (United States) 01 SEP 1998, 161/5 (2501-2508)
CODEN: JOIMA ISSN: 0022-1767
DOCUMENT TYPE: Journal; Article
LANGUAGE: ENGLISH SUMMARY LANGUAGE: ENGLISH
NUMBER OF REFERENCES: 49

In this study, an in vitro flow model and a blocking mAb to **P-**

selectin glycoprotein ligand-1 (**PSGL**-1) were used to define the role of **PSGL**-1 in monocyte attachment and rolling on E- and P-selectin and in attachment and accumulation on 6-h TNF-alpha-activated HUVEC. KPL1, an adhesion-blocking mAb directed against the tyrosine sulfate motif of **PSGL**-1, abolished monocyte- adhesive interactions with P-selectin, but only partially blocked monocyte interaction with E-selectin. Further analysis showed that on E-selectin, KPL1 blocked only secondary (i.e., monocyte/monocyte) interactions, but did not block primary (i.e., monocyte/E-selectin) interactions, with secondary adhesion accounting for 90% of the total adhesive interactions on either E- or P-selectin. On cytokine-activated HUVEC, monocytes initially attached and formed linear strings of adherent cells, which involved both primary and secondary adhesion. **PSGL**-1 or L-selectin mAb reduced string formation, and the combination of **PSGL**-1 and L-selectin mAb prevented monocyte strings and inhibited 86% of accumulation. Monocyte attachment and rolling on purified adherent monocytes were also critically dependent on **PSGL**-1 on the adherent monocytes. These studies document that secondary interactions between monocytes, mediated by **PSGL**-1, are crucial for monocyte initial attachment, rolling, and accumulation on activated endothelium under laminar shear flow.

2/7/13 (Item 1 from file: 399)
DIALOG(R)File 399:CA SEARCH(R)

133280563 CA: 133(20)280563a PATENT
Human antibodies that bind human IL-12 and methods for producing
INVENTOR(AUTHOR): Salfeld, Jochen G.; Roguska, Michael; Paskind, Michael; Banerjee, Subhashis; Tracey, Daniel E.; White, Michael; Kaymakcalan, Zehra; Labkovsky, Boris; Sakorafas, Paul; Friedrich, Stuart; Myles, Angela; Veldman, Geertruida M.; Venturini, Amy; Warne, Nicholas W.; Widom, Angela; Elvin, John G.; Duncan, Alexander R.; Derbyshire, Elaine J.; Carmen, Sara; Smith, Stephen; Holtet, Thor Las; Du, Fou Sarah L.
LOCATION: Germany,
ASSIGNEE: Basf A.-G.; Genetics Institute Inc.; et al.
PATENT: PCT International ; WO 200056772 A1 DATE: 20000928
APPLICATION: WO 2000US7946 (20000324) *US PV126603 (19990325)
PAGES: 377 pp. CODEN: PIXXD2 LANGUAGE: English CLASS: C07K-016/24A; C12N-015/13B; C12N-015/63B; C12N-005/10B; C07K-016/00B; A61K-039/395B; G01N-033/577B; C12P-021/08B; A61P-043/00B DESIGNATED COUNTRIES: AE; AG; AL ; AM; AT; AU; AZ; BA; BB; BG; BR; BY; CA; CH; CN; CR; CU; CZ; DE; DK; DM; DZ; EE; ES; FI; GB; GD; GE; GH; GM; HR; HU; ID; IL; IN; IS; JP; KE; KG; KP; KR; KZ; LC; LK; LR; LS; LT; LU; LV; MA; MD; MG; MK; MN; MW; MX; NO; NZ; PL; PT; RO; RU; SD; SE; SG; SI; SK; SL; TJ; TM; TR; TT; TZ; UA; UG; US; UZ; VN; YU; ZA; ZW; AM; AZ; BY; KG; KZ; MD; RU; TJ; TM DESIGNATED REGIONAL: GH; GM ; KE; LS; MW; SD; SL; SZ; TZ; UG; ZW; AT; BE; CH; CY; DE; DK; ES; FI; FR; GB; GR; IE; IT; LU; MC; NL; PT; SE; BF; BJ; CF; CG; CI; CM; GA; GN; GW; ML; MR; NE; SN; TD; TG
SECTION:
CA215003 Immunochemistry
CA203XXX Biochemical Genetics
IDENTIFIERS: human antibody interleukin 12 autoimmune disease, inflammation recombinant antibody human interleukin 12
DESCRIPTORS:
Immunoglobulins...
A; recombinant human antibodies that bind human IL-12 for treatment of autoimmune diseases and inflammatory diseases
Respiratory distress syndrome...
adult; recombinant human antibodies that bind human IL-12 for treatment of autoimmune diseases and inflammatory diseases
Interleukin 2 receptors...
.alpha.-chain; recombinant human antibodies that bind human IL-12 for treatment of autoimmune diseases and inflammatory diseases
Spinal column...
ankylosing spondylitis; recombinant human antibodies that bind human

IL-12 for treatment of autoimmune diseases and inflammatory diseases
Transforming growth factors...
.beta.-; recombinant human antibodies that bind human IL-12 for treatment of autoimmune diseases and inflammatory diseases
Interferons...
.beta.1, .beta.1a and .beta.1b; recombinant human antibodies that bind human IL-12 for treatment of autoimmune diseases and inflammatory diseases
Phytohemagglutinins...
blast proliferation assay; recombinant human antibodies that bind human IL-12 for treatment of autoimmune diseases and inflammatory diseases
Drug delivery systems...
carriers; recombinant human antibodies that bind human IL-12 for treatment of autoimmune diseases and inflammatory diseases
Antigens...
CD90; recombinant human antibodies that bind human IL-12 for treatment of autoimmune diseases and inflammatory diseases
Fatigue,biological...
chronic fatigue syndrome; recombinant human antibodies that bind human IL-12 for treatment of autoimmune diseases and inflammatory diseases
Immunoglobulins...
conjugates; recombinant human antibodies that bind human IL-12 for treatment of autoimmune diseases and inflammatory diseases
Intestine,disease...
Crohn's; recombinant human antibodies that bind human IL-12 for treatment of autoimmune diseases and inflammatory diseases
Anti-inflammatory agents...
cytokine; recombinant human antibodies that bind human IL-12 for treatment of autoimmune diseases and inflammatory diseases
Immunity...
disorder, acute and chronic; recombinant human antibodies that bind human IL-12 for treatment of autoimmune diseases and inflammatory diseases
Blood coagulation...
disseminated intravascular; recombinant human antibodies that bind human IL-12 for treatment of autoimmune diseases and inflammatory diseases
Immunoglobulins...
E; recombinant human antibodies that bind human IL-12 for treatment of autoimmune diseases and inflammatory diseases
Cytokines...
EMAP-II or endothelial-monocyte-activating polypeptide II; recombinant human antibodies that bind human IL-12 for treatment of autoimmune diseases and inflammatory diseases
Heart,disease...
failure; recombinant human antibodies that bind human IL-12 for treatment of autoimmune diseases and inflammatory diseases
Lung,disease...
fibrosis; recombinant human antibodies that bind human IL-12 for treatment of autoimmune diseases and inflammatory diseases
Immunoglobulins...
fragments; recombinant human antibodies that bind human IL-12 for treatment of autoimmune diseases and inflammatory diseases
Transplant and Transplantation...
graft-vs.-host reaction; recombinant human antibodies that bind human IL-12 for treatment of autoimmune diseases and inflammatory diseases
Immunoglobulins...
G1; recombinant human antibodies that bind human IL-12 for treatment of autoimmune diseases and inflammatory diseases
Immunoglobulins...
G2; recombinant human antibodies that bind human IL-12 for treatment of autoimmune diseases and inflammatory diseases
Immunoglobulins...
G3; recombinant human antibodies that bind human IL-12 for treatment of autoimmune diseases and inflammatory diseases
Immunoglobulins...

G4; recombinant human antibodies that bind human IL-12 for treatment of autoimmune diseases and inflammatory diseases
Immunoglobulins...
heavy chains; recombinant human antibodies that bind human IL-12 for treatment of autoimmune diseases and inflammatory diseases
Anemia(disease)...
hemolytic; recombinant human antibodies that bind human IL-12 for treatment of autoimmune diseases and inflammatory diseases
Purpura(disease)...
Henoch-Schoenlein's; recombinant human antibodies that bind human IL-12 for treatment of autoimmune diseases and inflammatory diseases
Nervous system...
Huntington's chorea; recombinant human antibodies that bind human IL-12 for treatment of autoimmune diseases and inflammatory diseases
Tumor necrosis factor receptors...
Ig conjugates; recombinant human antibodies that bind human IL-12 for treatment of autoimmune diseases and inflammatory diseases
Heart,disease...
infarction; recombinant human antibodies that bind human IL-12 for treatment of autoimmune diseases and inflammatory diseases
Parasite...
infection; recombinant human antibodies that bind human IL-12 for treatment of autoimmune diseases and inflammatory diseases
Cytokines...
inflammatory, anti-; recombinant human antibodies that bind human IL-12 for treatment of autoimmune diseases and inflammatory diseases
Intestine,disease...
inflammatory; recombinant human antibodies that bind human IL-12 for treatment of autoimmune diseases and inflammatory diseases
Complement... Signal transduction,biological... Thromboxanes...
inhibitors; recombinant human antibodies that bind human IL-12 for treatment of autoimmune diseases and inflammatory diseases
Drug delivery systems...
injections, i.v.; recombinant human antibodies that bind human IL-12 for treatment of autoimmune diseases and inflammatory diseases
Diabetes mellitus...
insulin-dependent; recombinant human antibodies that bind human IL-12 for treatment of autoimmune diseases and inflammatory diseases
Baboon... Chimpanzee... Macaca irus... Macaca mulatta... Marmoset...
Primate...
interleukin 12; recombinant human antibodies that bind human IL-12 for treatment of autoimmune diseases and inflammatory diseases
Rheumatoid arthritis...
juvenile; recombinant human antibodies that bind human IL-12 for treatment of autoimmune diseases and inflammatory diseases
Blood vessel,disease...
Kawasaki; recombinant human antibodies that bind human IL-12 for treatment of autoimmune diseases and inflammatory diseases
Immunoglobulins...
light chains; recombinant human antibodies that bind human IL-12 for treatment of autoimmune diseases and inflammatory diseases
Immunoglobulins...
M; recombinant human antibodies that bind human IL-12 for treatment of autoimmune diseases and inflammatory diseases
Antibodies...
monoclonal; recombinant human antibodies that bind human IL-12 for treatment of autoimmune diseases and inflammatory diseases
Spinal cord...
myelitis, acute transverse; recombinant human antibodies that bind human IL-12 for treatment of autoimmune diseases and inflammatory diseases
Kidney,disease...
nephrotic syndrome; recombinant human antibodies that bind human IL-12 for treatment of autoimmune diseases and inflammatory diseases
Antibodies...
neutralizing; recombinant human antibodies that bind human IL-12 for

treatment of autoimmune diseases and inflammatory diseases
Anti-inflammatory agents...
nonsteroidal; recombinant human antibodies that bind human IL-12 for treatment of autoimmune diseases and inflammatory diseases
Selectins...
P-, glycoprotein ligand; recombinant human antibodies that bind human IL-12 for treatment of autoimmune diseases and inflammatory diseases
Glycoproteins,specific or class...
p-selectin glycoprotein ligand; recombinant human antibodies that bind human IL-12 for treatment of autoimmune diseases and inflammatory diseases
Artery,disease...
periarteritis nodosa; recombinant human antibodies that bind human IL-12 for treatment of autoimmune diseases and inflammatory diseases
Bioassay...
phytohemagglutinin blast proliferation assay; recombinant human antibodies that bind human IL-12 for treatment of autoimmune diseases and inflammatory diseases
Biliary tract...
primary biliary cirrhosis; recombinant human antibodies that bind human IL-12 for treatment of autoimmune diseases and inflammatory diseases
Arthritis...
psoriatic arthritis; recombinant human antibodies that bind human IL-12 for treatment of autoimmune diseases and inflammatory diseases
(etc.)...
CAS REGISTRY NUMBERS:
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297777-31-2 297777-32-3 297777-33-4 297777-34-5 297777-35-6 amino acid sequence; recombinant human antibodies that bind human IL-12 for treatment of autoimmune diseases and inflammatory diseases
28088-64-4D analogs, recombinant human antibodies that bind human IL-12 for treatment of autoimmune diseases and inflammatory diseases
58-61-7 biological studies, agonists; recombinant human antibodies that bind human IL-12 for treatment of autoimmune diseases and inflammatory diseases
7782-44-7 biological studies, hyperbaric; recombinant human antibodies that bind human IL-12 for treatment of autoimmune diseases and inflammatory diseases
110-86-1D imidazole compds., recombinant human antibodies that bind human IL-12 for treatment of autoimmune diseases and inflammatory diseases
80449-02-1 142243-02-5 inhibitor; recombinant human antibodies that bind human IL-12 for treatment of autoimmune diseases and inflammatory diseases
9004-06-2 9015-82-1 9025-82-5 9029-60-1 122191-40-6 151769-16-3 inhibitors; recombinant human antibodies that bind human IL-12 for treatment of autoimmune diseases and inflammatory diseases
9036-21-9 IV, inhibitor; recombinant human antibodies that bind human IL-12 for treatment of autoimmune diseases and inflammatory diseases
288-32-4D pyridinyl compds., recombinant human antibodies that bind human IL-12 for treatment of autoimmune diseases and inflammatory diseases
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298200-43-8 298200-44-9 298200-45-0 298200-46-1 298200-47-2
298200-48-3 298200-49-4 298200-50-7 recombinant human antibodies that bind human IL-12 for treatment of autoimmune diseases and inflammatory diseases

226983-90-0 226984-98-1 297782-45-7 297782-46-8 297782-47-9
297782-48-0 297782-49-1 297782-50-4 297782-51-5 unclaimed nucleotide sequence; human antibodies that bind human IL-12 and methods for producing

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297737-41-8 297737-42-9 297737-43-0 297737-44-1 297737-45-2
297737-46-3 297737-47-4 297737-48-5 297737-49-6 297737-50-9
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297737-66-7 297737-67-8 297737-68-9 297737-69-0 297737-70-3
297737-71-4 297737-72-5 297737-73-6 297737-74-7 297737-75-8
297737-76-9 297737-77-0 297737-78-1 297737-79-2 297737-80-5
297737-81-6 297737-82-7 297737-83-8 297737-84-9 297737-85-0
297737-86-1 297737-87-2 297737-88-3 297737-89-4 297737-90-7
297737-91-8 297737-92-9 297737-93-0 297737-94-1 297737-95-2
297737-96-3 297737-97-4 297737-98-5 297737-99-6 297738-00-2
297738-01-3 297738-02-4 297738-03-5 297738-04-6 unclaimed sequence; human antibodies that bind human IL-12 and methods for producing

2/7/14 (Item 2 from file: 399)
DIALOG(R)File 399:CA SEARCH(R)

124135703 CA: 124(11)135703f PATENT
Method using agents inhibiting interaction between P-selectin??? and a P-selectin ligand for treating and preventing atherosclerosis
INVENTOR(AUTHOR): Wagner, Denisa D.; Johnson, Robert C.
LOCATION: USA
ASSIGNEE: Center for Blood Research, Inc.
PATENT: PCT International ; WO 9533484 A1 DATE: 951214
APPLICATION: WO 95US6940 (950601) *US 253663 (940603) *US 377798 (950124)
PAGES: 35 pp. CODEN: PIXXD2 LANGUAGE: English CLASS: A61K-039/395A; A61K-038/02B; A61K-038/16B; A61K-031/70B DESIGNATED COUNTRIES: CA; JP
DESIGNATED REGIONAL: AT; BE; CH; DE; DK; ES; FR; GB; GR; IE; IT; LU; MC; NL; PT; SE
SECTION:
CA201008 Pharmacology
IDENTIFIERS: P selectin ligand inhibition atherosclerosis treatment
DESCRIPTORS:
Plant...

agents derived from plant ext. for inhibiting interaction between P-selectin and P-selectin ligand for treating and preventing atherosclerosis
Heart,disease, restenosis...
agents for inhibiting interaction between P-selectin and P-selectin ligand for treating and preventing atherosclerosis
Blood platelet... Eosinophil... Leukocyte... Lymphocyte,natural killer cell ... Monocyte... Neutrophil...
agents inhibiting interaction between cellular P-selectin and P-selectin ligand for treating and preventing atherosclerosis
Antiarteriosclerotics,antiatherosclerotics... Blood-group substances,Lea, sialyl... Blood-group substances,Lex, sialyl... Carbohydrates and Sugars,biological studies... Glycoproteins,biological studies... Glycoproteins,specific or class, PSGL-1 (P-selectin glycoprotein ligand-1) ... Ligands... Receptors,P-selectins...
agents inhibiting interaction between P-selectin and P-selectin ligand for treating and preventing atherosclerosis
Lymphocyte,T-cell...
CD4+ and CD8+; agents inhibiting interaction between cellular P-selectin and P-selectin ligand for treating and preventing atherosclerosis
Artery,endothelium...
cell; agents inhibiting interaction between cellular P-selectin and P-selectin ligand for treating and preventing atherosclerosis
Antibodies... Antibodies,monoclonal... Peptides,biological studies... Proteins,biological studies... Sulfatides...
inhibitory; agents inhibiting interaction between P-selectin and P-selectin ligand for treating and preventing atherosclerosis
Lysosome...
membrane, glycoproteins; agents inhibiting interaction between P-selectin and P-selectin ligand for treating and preventing atherosclerosis
Ligands...
P-selectin, 160 kD monospecific; agents inhibiting interaction between P-selectin and P-selectin ligand for treating and preventing atherosclerosis
Blood-group substances,Lex, sialyl...
pentasaccharide; agents inhibiting interaction between P-selectin and P-selectin ligand for treating and preventing atherosclerosis
Mucopolysaccharides,lactosaminoglycans,biological studies...
poly-; agents inhibiting interaction between P-selectin and P-selectin ligand for treating and preventing atherosclerosis
Venoms...
snake; agents derived from snake venom for inhibiting interaction between P-selectin and P-selectin ligand for treating and preventing atherosclerosis
Snake...
venom; agents derived from snake venom for inhibiting interaction between P-selectin and P-selectin ligand for treating and preventing atherosclerosis
Carbohydrates and Sugars,biological studies...
2,6-sialic acid-contg.; agents inhibiting interaction between P-selectin and P-selectin ligand for treating and preventing atherosclerosis
Blood-group substances,Lex...
3'-O-sulfate; agents inhibiting interaction between P-selectin and P-selectin ligand for treating and preventing atherosclerosis
CAS REGISTRY NUMBERS:
9005-49-6D oligosaccharides, agents inhibiting interaction between P-selectin and P-selectin ligand for treating and preventing atherosclerosis

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? s (psgl? or p(w)selectin(w)glycoprotein(w)ligand) and (restenosis)

          746  PSGL?
      3756683  P
        24823  SELECTIN
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          250085  GLYCOPROTEIN
          293792  LIGAND
             739  P(W)SELECTIN(W)GLYCOPROTEIN(W)LIGAND
           24623  RESTENOSIS
      S3      13  (PSGL? OR P(W)SELECTIN(W)GLYCOPROTEIN(W)LIGAND) AND
                  (RESTENOSIS)
? rd s3

...completed examining records
      S4       9  RD S3 (unique items)
? t s4/7/all


 4/7/1     (Item 1 from file: 5)
DIALOG(R)File   5:Biosis Previews(R)
(c) 2001 BIOSIS. All rts. reserv.

13205270   BIOSIS NO.: 200100412419
Prevention of intimal hyperplasia with recombinant soluble P-
  selectin glycoprotein ligand-immunoglobulin in the
  porcine coronary artery balloon injury model.
AUTHOR: Wang Kai; Zhou Zhongmin; Zhou Xiaorong; Tarakji Khaldoun; Topol
  Eric J; Lincoff A Michael(a)
AUTHOR ADDRESS: (a)Cardiology Department, Cleveland Clinic Foundation, 9500
  Euclid Ave., F25, Cleveland, OH, 44195: lincofa@ccf.org**USA
JOURNAL: Journal of the American College of Cardiology  38 (2):p577-582
August, 2001
MEDIUM: print
ISSN: 0735-1097
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English
SUMMARY LANGUAGE: English

ABSTRACT: OBJECTIVES: The role of P-selectin in the process of
  restenosis was evaluated using a recombinant immunoglobulin (Ig)
  chimera form of its ligand, soluble P-selectin
  glycoprotein ligand-Ig (rPSGL-Ig), as a competitive inhibitor
  for the natural ligand on leukocytes. BACKGROUND: Inflammation and
  coagulation activation after vascular injury may be an important factor
  in the development of restenosis. P-selectin has been shown to
  mediate leukocyte-endothelium and leukocyte-platelet interaction. These
  interactions are mediated through binding of P-selectin to P-
  selectin glycoprotein ligand-1 (PSGL-1) located
  on the surface of leukocytes. METHODS: Balloon injury was induced in the
  left anterior descending and right coronary arteries of 16 pigs at a
  balloon/artery diameter ratio of 1.5:1. Either rPSGL-Ig (1 mg/kg) or
  saline was randomly administered 15 min before balloon injury as an
  intravenous bolus. Four weeks after injury, morphometric analysis,
  immunohistochemistry and histological evaluation were performed on
  injured arterial segments. RESULTS: Increased luminal area was found in
  the rPSGL-Ig group compared with the placebo group (1.63+-0.57 mm2 vs.
  1.26+-0.32 mm2, p=0.044) owing to significantly reduced neointimal
  hyperplasia (cross-sectional area, 0.46+-0.45 mm2 vs. 0.13+-0.11 mm2,
  p=0.013). Immunohistochemistry and histological evaluation showed a
  significant decrease in the presence of tumor necrosis factor-alpha,
  interleukin-1 beta, and infiltration of macrophages in the injured vessel
  segments in the rPSGL-Lg group. CONCLUSIONS: P-selectin antagonism using
  rPSGL-Ig decreases neointimal hyperplasia following balloon injury, by
  inhibiting the inflammatory and thrombotic responses at the site of
  balloon injury, which appears to play a pivotal role in the pathogenesis
  of restenosis.

 4/7/2     (Item 2 from file: 5)
DIALOG(R)File   5:Biosis Previews(R)
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13115159 BIOSIS NO.: 200100322308
Alterations in platelet thrombus formation, leukocyte recruitment, and intimal hyperplasia in P-selectin-deficient mice after transluminal femoral artery injury.
AUTHOR: Smyth Susan S(a); Reis Ernane D; Fallon John T; Gordon Ron; Coller Barry S(a)
AUTHOR ADDRESS: (a)Medicine, Mount Sinai School of Medicine, New York, NY** USA
JOURNAL: Blood 96 (11 Part 1):p813a November 16, 2000
MEDIUM: print
CONFERENCE/MEETING: 42nd Annual Meeting of the American Society of Hematology San Francisco, California, USA December 01-05, 2000
SPONSOR: American Society of Hematology
ISSN: 0006-4971
RECORD TYPE: Abstract
LANGUAGE: English
SUMMARY LANGUAGE: English

ABSTRACT: Platelet activation at the site of vascular injury, such as occurs after atherosclerotic plaque rupture or following percutaneous intervention, results in platelet-neutrophil interactions, which contribute to local thrombosis, downstream microcirculatory events, and systemic inflammation. The initial interaction of activated platelets with neutrophils is mediated by platelet P-selectin binding to neutrophil **PSGL**-1. To investigate the role of platelet-neutrophil interactions in response to arterial injury, we performed transluminal, wire-induced injury to the femoral artery of wild-type C57B1/6 mice (n=26) and P-selectin -/- mice (n=26). The femoral arteries of anesthesized male mice aged 8-10 weeks were injured by passing a 0.25 mm wire in the lumen of the femoral artery three times, and the mice were euthanized by perfusion fixation 1 h or 4 weeks after injury. 1 h after injury in wild-type mice, platelets were found adherent to the blood vessel wall and neutrophils were attached to the platelets. As viewed by TEM and SEM, the platelet layer varied between 1 and apprxeq3 platelets thick, and many of the platelets in contact with the wall were spread and at least partially degranulated. 1 h after injury of P-selectin -/- mice, the platelet layer appeared less compact and appeared to extend further into the lumen; moreover, more of the platelets appeared to retain their granules. There was a striking decrease in leukocyte attachment to the platelets. Four weeks after injury, the neointimal area in P-selectin -/- mice (2,100 +- 900 mum2) was significantly smaller than in the wild-type mice (10,200 +- 2,100 mum2) (p=0.004). These results indicate that P-selectin is required for neutrophil recruitment to platelets lining the vessel wall 1 h after injury and P-selectin deficiency protects mice from developing intimal hyperplasia 4 weeks after injury. Moreover, in the P-selectin -/- mice, the platelets depositing on the damaged vessel appeared to be less activated and to extend further into the lumen, suggesting that P-selectin may play a role in platelet activation and platelet thrombus formation. Our data support the possibility that antagonists to P-selectin may decrease intimal hyperplasia and clinical **restenosis** after percutaneous vascular interventions in humans.

4/7/3 (Item 3 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

12954079 BIOSIS NO.: 200100161228
Recombinant soluble **P-selectin glycoprotein ligand** -1-Ig reduces **restenosis** through inhibition of platelet-neutrophil adhesion after double angioplasty in swine.
AUTHOR: Bienvenu Jean-Guy; Tanguay Jean-Francois; Theoret Jean-Francois; Kumar Anjali; Schaub Robert G; Merhi Yahye(a)
AUTHOR ADDRESS: (a)Laboratory of Experimental Pathology, Montreal Heart

Institute, 5000 Belanger St E, Montreal, PQ, H1T 1C8: merhi@icm.umontreal.ca**Canada
JOURNAL: Circulation 103 (8):p1128-1134 February 27, 2001
MEDIUM: print
ISSN: 0009-7322
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English
SUMMARY LANGUAGE: English

ABSTRACT: Background: P-selectin mediates leukocyte recruitment to activated platelets and endothelium through its high-affinity receptor **P-selectin glycoprotein ligand**-1 (**PSGL**-1). Platelet and leukocyte activation and binding have been reported after coronary angioplasty and were correlated with **restenosis**. We investigated the effect of a recombinant soluble **PSGL**-1 (rPSGL-Ig) on the adhesion of platelets and neutrophils and the development of **restenosis** after double arterial injury. Methods and Results: Four weeks after angioplasty of both carotid arteries in pigs, a second angioplasty was performed at the same sites, 15 minutes after a single administration of vehicle or rPSGL-1 (1 mg/kg IV). Animals were euthanized 1 hour, 4 hours, 1 week, or 4 weeks later. Adhesion of autologous 51Cr-platelets and 111In-neutrophils was quantified and histological/morphometric analyses were performed. Although rPSGL-Ig did not affect adherence of these cells 1 hour after injury, it significantly reduced the adhesion of platelets (50% at 4 hours and 85% at 1 week) and neutrophils (50% at 4 hours and 78% at 1 week) to deeply injured arteries. At 4 weeks, the residual lumen was 63% larger in rPSGL-Ig-treated arteries as compared with control arteries (6.1+-0.6 versus 3.8+-0.1 mm2; P<0.002). The neointimal area was slightly reduced (0.5 in rPSGL-Ig versus 0.7 mm2 in control). The ratio of the external elastic lamina of injured to uninjured reference segments was >1 in treated arteries and <1 in control arteries. Conclusions: P-selectin antagonism with rPSGL-Ig inhibits early platelet/leukocyte adhesion on injured arteries and reduces **restenosis** through a positive impact on vascular remodeling. Hence, rPSGL-Ig may have potential in the prevention of **restenosis**.

4/7/4 (Item 4 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

12953911 BIOSIS NO.: 200100161060
rPSGL-Ig inhibits in-stent **restenosis** in porcine coronary arteries after double injury.
AUTHOR: Tanguay Jean-Francois(a); Geoffroy Pascale; Theoret Jean-Francois; Schaub Robert G; Kumar Anjali; Merhi Yahye
AUTHOR ADDRESS: (a)Montreal Heart Institute, Montreal, PQ**Canada
JOURNAL: Journal of the American College of Cardiology 37 (2 Supplement A):p28A February, 2001
MEDIUM: print
CONFERENCE/MEETING: 50th Annual Scientific Session of the American College of Cardiology Orlando, Florida, USA March 18-21, 2001
ISSN: 0735-1097
RECORD TYPE: Citation
LANGUAGE: English
SUMMARY LANGUAGE: English

4/7/5 (Item 5 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

12905204 BIOSIS NO.: 200100112353
Prevention of intimal hyperplasia with recombinant soluble **P**-

selectin glycoprotein ligand-Ig in the porcine coronary artery balloon injury model.
AUTHOR: Wang Kai(a); Zhou Zhong Min(a); Zhou Xiaorong(a); Lincoff A Michael (a)
AUTHOR ADDRESS: (a)Cleveland Clin Fdn, Cleveland, OH**USA
JOURNAL: Circulation 102 (18 Supplement):pII329-II330 October 31, 2000
MEDIUM: print
CONFERENCE/MEETING: Abstracts from Scientific Sessions 2000 New Orleans, Louisiana, USA November 12-15, 2000
ISSN: 0009-7322
RECORD TYPE: Citation
LANGUAGE: English
SUMMARY LANGUAGE: English

4/7/6 (Item 6 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

12839849 BIOSIS NO.: 200100046998
Effect of a recombinant soluble **P-selectin glycoprotein ligand**-1 on **restenosis** following arterial injury by repeat angioplasty in pigs.
AUTHOR: Bienvenu J G(a); Tanguay J F; Theoret J F; Kumar A; Schaub R G; Merhi Y
AUTHOR ADDRESS: (a)Montreal, PQ**Canada
JOURNAL: Canadian Journal of Cardiology 16 (Supplement F):p213F-214F September, 2000
MEDIUM: print
CONFERENCE/MEETING: 53rd Annual Meeting of the Canadian Cardiovascular Society Vancouver, British Columbia, Canada October 20-November 01, 2000
SPONSOR: Canadian Cardiovascular Society
ISSN: 0828-282X
RECORD TYPE: Citation
LANGUAGE: English
SUMMARY LANGUAGE: English

4/7/7 (Item 7 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

12411198 BIOSIS NO.: 200000164700
Effect of a recombinant soluble **P-Selectin Glycoprotein Ligand**-1 chimera on **restenosis** following arterial injury by repeat angioplasty in pigs.
AUTHOR: Bienvenu Jean-Guy(a); Tanguay Jean-Francois; Theoret Jean-Francois; Kumar Anjali; Schaub Robert G; Merhi Yahye
AUTHOR ADDRESS: (a)Montreal Heart Institute, Montreal, PQ**Canada
JOURNAL: Journal of the American College of Cardiology. 35 (2 suppl. A):p 16A Feb., 2000
CONFERENCE/MEETING: 29th Annual Scientific Session of the American College of Cardiology. Anaheim, California, USA March 12-15, 2000
ISSN: 0735-1097
RECORD TYPE: Citation
LANGUAGE: English
SUMMARY LANGUAGE: English

4/7/8 (Item 1 from file: 73)
DIALOG(R)File 73:EMBASE

07629695 EMBASE No: 1999099739
Recombinant soluble form of **PSGL**-1 accelerates thrombolysis and prevents reocclusion in a porcine model

Kumar A.; Villani M.P.; Patel U.K.; Keith J.C. Jr.; Schaub R.G.
Dr. A. Kumar, Preclinical R and D, Genetics Institute, Inc., One Burtt Rd, Andover, MA 01810 United States
AUTHOR EMAIL: akumar@genetics.com
Circulation (CIRCULATION) (United States) 16 MAR 1999, 99/10 (1363-1369)
CODEN: CIRCA ISSN: 0009-7322
DOCUMENT TYPE: Journal; Article
LANGUAGE: ENGLISH SUMMARY LANGUAGE: ENGLISH
NUMBER OF REFERENCES: 42

Background - We investigated whether administration of a soluble recombinant **P-selectin glycoprotein ligand**-1 chimera (rPSGL-Ig) in conjunction with thrombolytic therapy would enhance thrombolysis by preventing ongoing interactions of leukocytes with platelets and the injured arterial wall. Methods and Results - An occlusive thrombus was formed in an internal iliac artery of Yorkshire pigs by placement of a copper coil in the artery under fluoroscopic guidance. Pigs then received heparin and, 15 minutes later, either vehicle or rPSGL-Ig followed by infusion with 25 mg tissue plasminogen activator according to the 90-minute regimen. Blood flow through the artery was monitored by angiography and scored on a scale of 0 to 3. Lysis of the thrombus was accelerated by 70% in pigs treated with rPSGL- Ig 250 mug/kg compared with control (13.3+/-5.0 versus 44.4+/-13.3 minutes; n=9 each). Eight of 9 control pigs reoccluded in 13.8+/-16.9 minutes after the end of tissue plasminogen activator infusion, whereas no reocclusion was observed in 8 of 9 pigs in the rPSGL-Ig group. When the dose of rPSGL-Ig was increased to 500 mug/kg, time to lysis was shortened by 61% from control (18.0+/-8.4 versus 46.0+/-8.9 minutes). Reocclusion occurred in 6.0+/-15.2 minutes in control but not in any rPSGL-Ig-treated pig (n=5 each). In addition, near-normal flow (score 2 or 3) after thrombolysis was achieved 59% and 58% faster in the 2 rPSGL-Ig groups than in their respective controls. Conclusions - Inhibition of leukocyte accumulation at the site of thrombosis with rPSGL-Ig may represent a safe therapeutic intervention that could be important in accelerating thrombolysis, achieving optimal reperfusion, and reducing incidence of acute reocclusion.

4/7/9 (Item 1 from file: 399)
DIALOG(R)File 399:CA SEARCH(R)

124135703 CA: 124(11)135703f PATENT
Method using agents inhibiting interaction between P-selectin??? and a P-selectin ligand for treating and preventing atherosclerosis
INVENTOR(AUTHOR): Wagner, Denisa D.; Johnson, Robert C.
LOCATION: USA
ASSIGNEE: Center for Blood Research, Inc.
PATENT: PCT International ; WO 9533484 A1 DATE: 951214
APPLICATION: WO 95US6940 (950601) *US 253663 (940603) *US 377798 (950124)
PAGES: 35 pp. CODEN: PIXXD2 LANGUAGE: English CLASS: A61K-039/395A; A61K-038/02B; A61K-038/16B; A61K-031/70B DESIGNATED COUNTRIES: CA; JP
DESIGNATED REGIONAL: AT; BE; CH; DE; DK; ES; FR; GB; GR; IE; IT; LU; MC; NL; PT; SE
SECTION:
CA201008 Pharmacology
IDENTIFIERS: P selectin ligand inhibition atherosclerosis treatment
DESCRIPTORS:
Plant...
agents derived from plant ext. for inhibiting interaction between P-selectin and P-selectin ligand for treating and preventing atherosclerosis
Heart,disease, restenosis...
agents for inhibiting interaction between P-selectin and P-selectin ligand for treating and preventing atherosclerosis
Blood platelet... Eosinophil... Leukocyte... Lymphocyte,natural killer cell

... Monocyte... Neutrophil...
agents inhibiting interaction between cellular P-selectin and P-selectin ligand for treating and preventing atherosclerosis
Antiarteriosclerotics,antiatherosclerotics... Blood-group substances,Lea, sialyl... Blood-group substances,Lex, sialyl... Carbohydrates and Sugars,biological studies... Glycoproteins,biological studies... Glycoproteins,specific or class, PSGL-1 (P-selectin glycoprotein ligand-1) ... Ligands... Receptors,P-selectins...
agents inhibiting interaction between P-selectin and P-selectin ligand for treating and preventing atherosclerosis
Lymphocyte,T-cell...
CD4+ and CD8+; agents inhibiting interaction between cellular P-selectin and P-selectin ligand for treating and preventing atherosclerosis
Artery,endothelium...
cell; agents inhibiting interaction between cellular P-selectin and P-selectin ligand for treating and preventing atherosclerosis
Antibodies... Antibodies,monoclonal... Peptides,biological studies... Proteins,biological studies... Sulfatides...
inhibitory; agents inhibiting interaction between P-selectin and P-selectin ligand for treating and preventing atherosclerosis
Lysosome...
membrane, glycoproteins; agents inhibiting interaction between P-selectin and P-selectin ligand for treating and preventing atherosclerosis
Ligands...
P-selectin, 160 kD monospecific; agents inhibiting interaction between P-selectin and P-selectin ligand for treating and preventing atherosclerosis
Blood-group substances,Lex, sialyl...
pentasaccharide; agents inhibiting interaction between P-selectin and P-selectin ligand for treating and preventing atherosclerosis
Mucopolysaccharides,lactosaminoglycans,biological studies...
poly-; agents inhibiting interaction between P-selectin and P-selectin ligand for treating and preventing atherosclerosis
Venoms...
snake; agents derived from snake venom for inhibiting interaction between P-selectin and P-selectin ligand for treating and preventing atherosclerosis
Snake...
venom; agents derived from snake venom for inhibiting interaction between P-selectin and P-selectin ligand for treating and preventing atherosclerosis
Carbohydrates and Sugars,biological studies...
2,6-sialic acid-contg.; agents inhibiting interaction between P-selectin and P-selectin ligand for treating and preventing atherosclerosis
Blood-group substances,Lex...
3'-O-sulfate; agents inhibiting interaction between P-selectin and P-selectin ligand for treating and preventing atherosclerosis
CAS REGISTRY NUMBERS:
9005-49-6D oligosaccharides, agents inhibiting interaction between P-selectin and P-selectin ligand for treating and preventing atherosclerosis
? ds

Set	Items	Description
S1	25	(PSGL? OR P(W)SELECTIN(W)GLYCOPROTEIN(W)LIGAND) AND (ATHEROSCLER?)
S2	14	RD S1 (unique items)
S3	13	(PSGL? OR P(W)SELECTIN(W)GLYCOPROTEIN(W)LIGAND) AND (RESTENOSIS)
S4	9	RD S3 (unique items)

? s s2 and s4

```
Set     Items   Description
S1         25   (PSGL? OR P(W)SELECTIN(W)GLYCOPROTEIN(W)LIGAND) AND (ATHER-
             OSCLER?)
S2         14   RD S1 (unique items)
S3         13   (PSGL? OR P(W)SELECTIN(W)GLYCOPROTEIN(W)LIGAND) AND (RESTE-
             NOSIS)
S4          9   RD S3 (unique items)
S5          2   S2 AND S4
S6          2   RD S5 (unique items)
S7       3360   RESTENOSIS AND ATHEROSCLEROSIS
S8        495   S7 AND REVIEW?
S9        303   RESTENOSIS (20N) ATHEROSCLEROSIS AND REVIEW?
S10         0   S9 AND P(W)SELECTION?
S11         1   S9 AND P(W)SELECTIN?
? s (restenosis or atherosclerosis) and p(w)selectin?

          24623  RESTENOSIS
         136871  ATHEROSCLEROSIS
        3756683  P
          66839  SELECTIN?
           9434  P(W)SELECTIN?
     S12    511  (RESTENOSIS OR ATHEROSCLEROSIS) AND P(W)SELECTIN?
? s s12 and review?

            511  S12
        3019137  REVIEW?
     S13     27  S12 AND REVIEW?
? rd s13

...completed examining records
     S14     21  RD S13 (unique items)
? t s14/3/all


 14/3/1     (Item 1 from file: 5)
DIALOG(R)File   5:Biosis Previews(R)
(c) 2001 BIOSIS. All rts. reserv.

13218531   BIOSIS NO.: 200100425680
Early increase in levels of soluble inter-cellular adhesion molecule-1
  (sICAM-1): Potential risk factor for the acute coronary syndromes.
AUTHOR: O'Malley T; Ludlam C A; Riemermsa R A; Fox K A A(a)
AUTHOR ADDRESS: (a)Cardiovascular Research, Department of Medical and
  Radiological Sciences, Cardiology, The University of Edinburgh, Royal
  Infirmary of Edinburgh, Lauriston Place, Edinburgh, EH3 9YW**UK
JOURNAL: European Heart Journal  22 (14):p1226-1234 July, 2001
MEDIUM: print
ISSN: 0195-668X
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English
SUMMARY LANGUAGE: English


 14/3/2     (Item 2 from file: 5)
DIALOG(R)File   5:Biosis Previews(R)
(c) 2001 BIOSIS. All rts. reserv.
```

11352656 BIOSIS NO.: 199800133988
Platelet-leukocyte-cross-talk in diabetes mellitus.
AUTHOR: Tschoepe D(a); Rauch U; Schwippert B
AUTHOR ADDRESS: (a)Diabetes Research Inst. at Henirch Heine Univ., Cellular Haemostasis and Clinical Angiology Grou**Germany
JOURNAL: Hormone and Metabolic Research 29 (12):p631-635 Dec., 1997
ISSN: 0018-5043
DOCUMENT TYPE: Literature Review
RECORD TYPE: Abstract
LANGUAGE: English

14/3/3 (Item 3 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

09050005 BIOSIS NO.: 199497058375
Potential roles for oxidized phospholipids in inflammation and atherogenesis.
BOOK TITLE: **Atherosclerosis Reviews; Atherosclerosis:** Cellular interactions, growth factors, and lipids
AUTHOR: Prescott Stephen M(a); Patel Kamala D; Smiley Patricia L; Stafforini Diana M; Lorant Diane E; Zimmerman Guy A; McIntyre Thomas M
BOOK AUTHOR/EDITOR: Weber P C; Leaf A: Eds
AUTHOR ADDRESS: (a)Eccles Inst. Human Genet., Room 4220, Univ. Utah, Salt Lake City, UT 84112**USA
JOURNAL: Atherosclerosis Reviews 25p59-68 1993
BOOK PUBLISHER: Raven Press, 1185 Avenue of the Americas, New York, New York 10036-2806, USA
CONFERENCE/MEETING: Third Miles International Workshop on Atherosclerosis Stresa, Italy September 30-October 2, 1992
ISSN: 0362-1650 ISBN: 0-7817-0099-X
RECORD TYPE: Citation
LANGUAGE: English

14/3/4 (Item 1 from file: 73)
DIALOG(R)File 73:EMBASE

11358152 EMBASE No: 2001372385
The vascular-associated lymphoid tissue: A new site of local immunity
Millonig G.; Schwentner C.; Mueller P.; Mayerl C.; Wick G.
Dr. G. Wick, Inst. for Biomedical Aging Research, Austrian Academy of Sciences, Rennweg 10, 6020 Innsbruck Austria
AUTHOR EMAIL: georg.wick@uibk.ac.at
Current Opinion in Lipidology (CURR. OPIN. LIPIDOLOGY) (United Kingdom)
2001, 12/5 (547-553)
CODEN: COPLE ISSN: 0957-9672
DOCUMENT TYPE: Journal ; Review
LANGUAGE: ENGLISH SUMMARY LANGUAGE: ENGLISH
NUMBER OF REFERENCES: 48

14/3/5 (Item 2 from file: 73)
DIALOG(R)File 73:EMBASE

11344635 EMBASE No: 2001358926
Adhesion molecules and atherogenesis
Huo Y.; Ley K.
K. Ley, Department of Biomedical Engineering, University of Virginia, Health Science Center, Charlottesville, VA 22908 United States
Acta Physiologica Scandinavica (ACTA PHYSIOL. SCAND.) (United Kingdom)
2001, 173/1 (35-43)
CODEN: APSCA ISSN: 0001-6772

DOCUMENT TYPE: Journal ; Review
LANGUAGE: ENGLISH SUMMARY LANGUAGE: ENGLISH
NUMBER OF REFERENCES: 56

14/3/6 (Item 3 from file: 73)
DIALOG(R)File 73:EMBASE

11302989 EMBASE No: 2001317223
Adhesive interactions of leukocytes, platelets, and the vessel wall during hemostasis and inflammation
McEver R.P.
Dr. R.P. McEver, Warren Medical Research Institute, University of Oklahoma, Health Sciences Center, 825 N. E. 13th Street, Oklahoma City, OK 73104 United States
AUTHOR EMAIL: rodger-mcever@ouhsc.edu
Thrombosis and Haemostasis (THROMB. HAEMOST.) (Germany) 2001, 86/3 (746-756)
CODEN: THHAD ISSN: 0340-6245
DOCUMENT TYPE: Journal ; Review
LANGUAGE: ENGLISH
NUMBER OF REFERENCES: 149

14/3/7 (Item 4 from file: 73)
DIALOG(R)File 73:EMBASE

10888487 EMBASE No: 2000349489
Inhibition of cellular action of thrombin by N3-cyclopropyl-7-{[4-(1-methylethyl)phenyl]methyl}-7H-pyrrolo[3,2-f]quinazoline-1,3-diamine (SCH 79797), a nonpeptide thrombin receptor antagonist
Ahn H.-S.; Foster C.; Boykow G.; Stamford A.; Manna M.; Graziano M.
Dr. H.-S. Ahn, Department of CNS, CV Biological Research, Schering-Plough Research Institute, 2015 Galloping Hill Road, Kenilworth, NJ 07033-1300 United States
AUTHOR EMAIL: ho-sam.ahn@spcorp.com
Biochemical Pharmacology (BIOCHEM. PHARMACOL.) (United States) 15 NOV 2000, 60/10 (1425-1434)
CODEN: BCPCA ISSN: 0006-2952
PUBLISHER ITEM IDENTIFIER: S0006295200004603
DOCUMENT TYPE: Journal; Review
LANGUAGE: ENGLISH SUMMARY LANGUAGE: ENGLISH
NUMBER OF REFERENCES: 52

14/3/8 (Item 5 from file: 73)
DIALOG(R)File 73:EMBASE

10863398 EMBASE No: 2000345938
Roles of **P-selectin** in inflammation, neointimal formation, and vascular remodeling in balloon-injured rat carotid arteries
Hayashi S.-I.; Watanabe N.; Nakazawa K.; Suzuki J.; Tsushima K.; Tamatani T.; Sakamoto S.; Isobe M.
Dr. M. Isobe, Dept. of Cardiovascular Medicine, Tokyo Medical and Dental University, 1-5-45 Yushima, Bunkyo-ku, Tokyo 113-8519 Japan
AUTHOR EMAIL: isobemi.med3@med.tmd.ac.jp
Circulation (CIRCULATION) (United States) 03 OCT 2000, 102/14 (1710-1717)
CODEN: CIRCA ISSN: 0009-7322
DOCUMENT TYPE: Journal; Review
LANGUAGE: ENGLISH SUMMARY LANGUAGE: ENGLISH
NUMBER OF REFERENCES: 24

14/3/9 (Item 6 from file: 73)
DIALOG(R)File 73:EMBASE
(c) 2001 Elsevier Science B.V. All rts. reserv.

07291663 EMBASE No: 1998185085
Effects of viral activation of the vessel wall on inflammation and thrombosis
Vercellotti G.M.
Prof. G.M. Vercellotti, Univ. of Minnesota Medical School, Box 293 Mayo, 420 Delaware St SE, Minneapolis, MN 55105 United States
Blood Coagulation and Fibrinolysis (BLOOD COAGUL. FIBRINOLYSIS) (United Kingdom) 1998, 9/SUPPL.. 2 (S3-S6)
CODEN: BLFIE ISSN: 0957-5235
DOCUMENT TYPE: Journal; Review
LANGUAGE: ENGLISH SUMMARY LANGUAGE: ENGLISH
NUMBER OF REFERENCES: 24

14/3/10 (Item 7 from file: 73)
DIALOG(R)File 73:EMBASE
(c) 2001 Elsevier Science B.V. All rts. reserv.

07050505 EMBASE No: 1997332349
Thrombosis and **atherosclerosis**
Holvoet P.; Collen D.
P. Holvoet, Center Molecular Vascular Biology, University of Leuven, Campus Gasthuisberg, Herestraat 49, B-3000 Leuven Belgium
Current Opinion in Lipidology (CURR. OPIN. LIPIDOLOGY) (United Kingdom) 1997, 8/5 (320-328)
CODEN: COPLE ISSN: 0957-9672
DOCUMENT TYPE: Journal; Review
LANGUAGE: ENGLISH SUMMARY LANGUAGE: ENGLISH
NUMBER OF REFERENCES: 89

14/3/11 (Item 8 from file: 73)
DIALOG(R)File 73:EMBASE
(c) 2001 Elsevier Science B.V. All rts. reserv.

06849256 EMBASE No: 1997131844
Microcirculation, vitamin E and omega 3 fatty acids: An overview
Bruckner G.
G. Bruckner, Division Clinical Nutrition, Department of Clinical Sciences, University of Kentucky, Lexington, KY 40506-0080 United States
Advances in Experimental Medicine and Biology (ADV. EXP. MED. BIOL.) (United States) 1997, 415/- (195-208)
CODEN: AEMBA ISSN: 0065-2598
DOCUMENT TYPE: Journal; Review
LANGUAGE: ENGLISH SUMMARY LANGUAGE: ENGLISH
NUMBER OF REFERENCES: 104

14/3/12 (Item 9 from file: 73)
DIALOG(R)File 73:EMBASE
(c) 2001 Elsevier Science B.V. All rts. reserv.

06492983 EMBASE No: 1996159307
Inflammation as an early component of **atherosclerosis** and vascular damage: A role for **P-selectin** and platelet-activating factor
Prescott S.M.; McIntyre T.M.; Zimmerman G.A.; Stafforini D.M.
Program in Human Molecular Biology, Genetics Eccles Human Genetics Inst., University of Utah,Salt Lake City, UT 84112 United States
Japanese Circulation Journal (JPN. CIRC. J.) (Japan) 1996, 60/3 (137-141)
CODEN: JCIRA ISSN: 0047-1828

DOCUMENT TYPE: Journal; Review
LANGUAGE: ENGLISH

14/3/13 (Item 10 from file: 73)
DIALOG(R)File 73:EMBASE
(c) 2001 Elsevier Science B.V. All rts. reserv.

05383475 EMBASE No: 1993151574
Blood monocyte adhesion to vascular endothelial cells. Implication in vascular pathology
Dosquet C.; Wautier J.-L.
Lab de Biologie Vasculaire/, Cellulaire, Hopital Lariboisiere, 2 rue Ambroise Pare,75010 Paris France
Clinical Hemorheology (CLIN. HEMORHEOL.) (United States) 1992, 12/6 (817-829)
CODEN: CLHED ISSN: 0271-5198
DOCUMENT TYPE: Journal; Review
LANGUAGE: ENGLISH SUMMARY LANGUAGE: ENGLISH

14/3/14 (Item 11 from file: 73)
DIALOG(R)File 73:EMBASE
(c) 2001 Elsevier Science B.V. All rts. reserv.

05336497 EMBASE No: 1993104582
Platelet alpha-granules
Harrison P.; Cramer E.M.
Coagulation Research, Rayne Institute St, Thomas' Hospital,London SE1 7EH United Kingdom
Blood Reviews (BLOOD REV.) (United Kingdom) 1993, 7/1 (52-62)
CODEN: BLORE ISSN: 0268-960X
DOCUMENT TYPE: Journal; Review
LANGUAGE: ENGLISH SUMMARY LANGUAGE: ENGLISH

14/3/15 (Item 1 from file: 155)
DIALOG(R)File 155:MEDLINE(R)

11750624 21379944 PMID: 11487452
Platelet-endothelial interactions in **atherosclerosis.**
Sachais BS
Department of Pathology and Laboratory Medicine, University of Pennsylvania, 3620 Hamilton Walk, 207A John Morgan Building, Philadelphia, PA 19104, USA. sachais@mail.med.upenn.edu
Current atherosclerosis reports (United States) Sep 2001, 3 (5) p412-6, ISSN 1523-3804 Journal Code: DYL
Languages: ENGLISH
Document type: Journal Article; Review; Review, Tutorial
Record type: Completed

14/3/16 (Item 1 from file: 399)
DIALOG(R)File 399:CA SEARCH(R)
(c) 2001 AMERICAN CHEMICAL SOCIETY. All rts. reserv.

130151597 CA: 130(12)151597d JOURNAL
Tissue factor expression by monocytes: regulation and pathophysiological roles
AUTHOR(S): Osterud, B.
LOCATION: Department of Biochemistry, Institute of Medical Biology, University of Tromso, Tromso, Norway
JOURNAL: Blood Coagulation Fibrinolysis DATE: 1998 VOLUME: 9 NUMBER: Suppl. 1 PAGES: S9-S14 CODEN: BLFIE7 ISSN: 0957-5235 LANGUAGE: English
PUBLISHER: Lippincott-Raven Publishers

14/3/17 (Item 2 from file: 399)
DIALOG(R)File 399:CA SEARCH(R)
(c) 2001 AMERICAN CHEMICAL SOCIETY. All rts. reserv.

129120808 CA: 129(10)120808y JOURNAL
Atherosclerosis and activation of platelet and blood coagulation
AUTHOR(S): Komiyama, Yutaka; Takahashi, Hakuo
LOCATION: Dep. Clin. Sci. Lab. Med., Kansai Med. Univ., Moriguchi, Japan, 570-8507
JOURNAL: Rinsho Byori DATE: 1998 VOLUME: 46 NUMBER: 7 PAGES: 678-683
CODEN: RBYOAI ISSN: 0047-1860 LANGUAGE: Japanese PUBLISHER: Rinsho Byori Kankokai

14/3/18 (Item 3 from file: 399)
DIALOG(R)File 399:CA SEARCH(R)
(c) 2001 AMERICAN CHEMICAL SOCIETY. All rts. reserv.

129079783 CA: 129(7)79783w JOURNAL
Endothelial adhesion molecules in health and disease
AUTHOR(S): Cotran, R. S.; Mayadas-Norton, T.
LOCATION: Department of Pathology, Harvard Medical School, Boston, MA, 02115, USA
JOURNAL: Pathol. Biol. DATE: 1998 VOLUME: 46 NUMBER: 3 PAGES: 164-170
CODEN: PTBIAN ISSN: 0031-3009 LANGUAGE: English PUBLISHER: Expansion Scientifique Publications

14/3/19 (Item 4 from file: 399)
DIALOG(R)File 399:CA SEARCH(R)
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128113469 CA: 128(10)113469q JOURNAL
Role of adhesion molecules in atherogenesis
AUTHOR(S): Yoshida, Masayuki
LOCATION: Med. Res. Inst., Tokyo Med. Dent. Univ., Tokyo, Japan, 101
JOURNAL: Domyaku Koka DATE: 1997 VOLUME: 25 NUMBER: 3 PAGES: 113-119
CODEN: DOMKDM ISSN: 0386-2682 LANGUAGE: Japanese PUBLISHER: Nippon Domyaku Koka Gakkai

14/3/20 (Item 5 from file: 399)
DIALOG(R)File 399:CA SEARCH(R)
(c) 2001 AMERICAN CHEMICAL SOCIETY. All rts. reserv.

124339381 CA: 124(25)339381j JOURNAL
Adhesion molecules influencing atherosclerosis
AUTHOR(S): Tschoepe, D.
LOCATION: Diabetes Research Institute, Heinrich Heine University, Duesseldorf, Germany, 40225
JOURNAL: Diabetes Res. Clin. Pract. DATE: 1996 VOLUME: 30 NUMBER: Suppl. PAGES: S19-S24 CODEN: DRCPE9 ISSN: 0168-8227 LANGUAGE: English

14/3/21 (Item 6 from file: 399)
DIALOG(R)File 399:CA SEARCH(R)
(c) 2001 AMERICAN CHEMICAL SOCIETY. All rts. reserv.

118036695 CA: 118(5)36695z JOURNAL
Viral activation of the coagulation cascade
AUTHOR(S): Etingin, Orli R.; Silverstein, Roy L.; Hajjar, David P.
LOCATION: Med. Coll., Cornell Univ., New York, NY, 10021, USA
JOURNAL: Semin. Virol. DATE: 1992 VOLUME: 3 NUMBER: 2 PAGES: 125-33
CODEN: SEVIEL ISSN: 1044-5773 LANGUAGE: English
? ds

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Set     Items   Description
S1         25   (PSGL? OR P(W)SELECTIN(W)GLYCOPROTEIN(W)LIGAND) AND (ATHER-
             OSCLER?)
S2         14   RD S1 (unique items)
S3         13   (PSGL? OR P(W)SELECTIN(W)GLYCOPROTEIN(W)LIGAND) AND (RESTE-
             NOSIS)
S4          9   RD S3 (unique items)
S5          2   S2 AND S4
S6          2   RD S5 (unique items)
S7       3360   RESTENOSIS AND ATHEROSCLEROSIS
S8        495   S7 AND REVIEW?
S9        303   RESTENOSIS (20N) ATHEROSCLEROSIS AND REVIEW?
S10         0   S9 AND P(W)SELECTION?
S11         1   S9 AND P(W)SELECTIN?
S12       511   (RESTENOSIS OR ATHEROSCLEROSIS) AND P(W)SELECTIN?
S13        27   S12 AND REVIEW?
S14        21   RD S13 (unique items)
? s s12 and py=1994

           511  S12
       1920496  PY=1994
     S15    18  S12 AND PY=1994
? rd s15

...completed examining records
     S16    13  RD S15 (unique items)
? t s16/3/all


 16/3/1     (Item 1 from file: 5)
DIALOG(R)File   5:Biosis Previews(R)
(c) 2001 BIOSIS. All rts. reserv.

09627100   BIOSIS NO.: 199598082018
Inhibition of platelet activity by S-nitrosoglutathione during coronary
  angioplasty.
AUTHOR: Langford E J; Brown A S; Wainwright R J; Debelder A J; Thomas M R;
  Smith R E A; Radomski M W; Martin J F(a); Moncada S
AUTHOR ADDRESS: (a)Cardiol. Dep., King's Coll. Hosp., London**UK
JOURNAL: Lancet (North American Edition)  344 (8935):p1458-1460 1994
ISSN: 0099-5355
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English


 16/3/2     (Item 2 from file: 5)
DIALOG(R)File   5:Biosis Previews(R)
(c) 2001 BIOSIS. All rts. reserv.

09566998   BIOSIS NO.: 199598021916
Role of P-selectin in animal models of thrombosis and
  restenosis.
AUTHOR: Shebuski Ronald J; Humphrey William R; Simmons Carol A; Hoover
  Jennifer L; Degraaf Garry L; Geng Jian G; Toombs Christopher F; Anderson
  Donald C
AUTHOR ADDRESS:  Upjohn Lab., Kalamazoo, MI**USA
JOURNAL: Circulation  90 (4 PART 2):pI142 1994
CONFERENCE/MEETING: 67th Scientific Sessions of the American Heart
Association  Dallas, Texas, USA  November 14-17, 1994
ISSN: 0009-7322
RECORD TYPE: Citation
LANGUAGE: English
```

16/3/3 (Item 3 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)
(c) 2001 BIOSIS. All rts. reserv.

09359869 BIOSIS NO.: 199497368239
Increase in the adhesion molecule **P-selectin** in endothelium overlying atherosclerotic plaques: Coexpression with intercellular adhesion molecule-1.
AUTHOR: Johnson-Tidey Ruth R; McGregor John L; Taylor Peter R; Poston Robin N(a)
AUTHOR ADDRESS: (a)Dep. Experimental Pathology, UMDS, Med. Sch., 4th Floor, Guy's Hosp., London Bridge, London SE1 **UK
JOURNAL: American Journal of Pathology 144 (5):p952-961 **1994**
ISSN: 0002-9440
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English

16/3/4 (Item 4 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)
(c) 2001 BIOSIS. All rts. reserv.

09226788 BIOSIS NO.: 199497235158
The protective role of high-density lipoprotein on oxidized-low-density-lipoprotein-induced U937/endothelial cell interactions.
AUTHOR: Maier Jeanette Anne Marie(a); Barenghi Livia; Pagani Franco; Bradamante Silvia; Comi Paola; Ragnotti Giovanni
AUTHOR ADDRESS: (a)Dipartimento di Scienze Tecnol. Biomediche-Ospedale San Raffaele, Via Olgettina 58, I-20132 Mila**Italy
JOURNAL: European Journal of Biochemistry 221 (1):p35-41 **1994**
ISSN: 0014-2956
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English

16/3/5 (Item 5 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)
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09156371 BIOSIS NO.: 199497164741
Purification of a novel cobra venom protease that cleaves the Von Willebrand factor receptor on human platelets and the **P-selectin** receptor on neutrophils.
AUTHOR: Andrews Robert K; Ward Christopher M; Dunlop Lindsay C; Berndt Michael C
AUTHOR ADDRESS: Vascular Biol. Lab., Baker Med. Research Inst., Prahran 3181**Australia
JOURNAL: Journal of Cellular Biochemistry Supplement 0 (18 PART A):p294 **1994**
CONFERENCE/MEETING: Keystone Symposium on Inflammation, Growth Regulatory Molecules and Atherosclerosis Keystone, Colorado, USA January 16-23, 1994
ISSN: 0733-1959
RECORD TYPE: Citation
LANGUAGE: English

16/3/6 (Item 6 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)
(c) 2001 BIOSIS. All rts. reserv.

09156365 BIOSIS NO.: 199497164735
Expression cloning of a functional glycoprotein ligand for **P-**

selectin.
AUTHOR: Sako Dianne(a); Chang Xiao-Jia(a); Barone Karen M(a); Vachino Gloria(a); Shaw Gray(a); Veldman Trudi M(a); Bean Kevin M(a); Ahern Tim J (a); Furie Bruce; et al
AUTHOR ADDRESS: (a)Genetics Inst. Inc., 87 Cambridge Park Drive, Cambridge, MA 02140**USA
JOURNAL: Journal of Cellular Biochemistry Supplement 0 (18 PART A):p293
1994
CONFERENCE/MEETING: Keystone Symposium on Inflammation, Growth Regulatory Molecules and Atherosclerosis Keystone, Colorado, USA January 16-23, 1994
ISSN: 0733-1959
RECORD TYPE: Citation
LANGUAGE: English

16/3/7 (Item 7 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

09156358 BIOSIS NO.: 199497164728
Increase in **P-selectin** in the endothelium overlying human atherosclerotic plaques: Coexpression with ICAM-1.
AUTHOR: Johnson-Tidey Ruth R; Poston Robin N
AUTHOR ADDRESS: Dep. Experimental Pathol., UMDS, Guy's Hosp., London SE1 9RT**UK
JOURNAL: Journal of Cellular Biochemistry Supplement 0 (18 PART A):p291
1994
CONFERENCE/MEETING: Keystone Symposium on Inflammation, Growth Regulatory Molecules and Atherosclerosis Keystone, Colorado, USA January 16-23, 1994
ISSN: 0733-1959
RECORD TYPE: Citation
LANGUAGE: English

16/3/8 (Item 8 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

09156353 BIOSIS NO.: 199497164723
Structure/function studies of **P-selectin** glycoprotein ligand.
AUTHOR: Barone Karen M; Pittman Deborah; Shaw Gray
AUTHOR ADDRESS: Genetics Inst. Inc., 87 Cambridge Park Drive, Cambridge, MA 02140**USA
JOURNAL: Journal of Cellular Biochemistry Supplement 0 (18 PART A):p290
1994
CONFERENCE/MEETING: Keystone Symposium on Inflammation, Growth Regulatory Molecules and Atherosclerosis Keystone, Colorado, USA January 16-23, 1994
ISSN: 0733-1959
RECORD TYPE: Citation
LANGUAGE: English

16/3/9 (Item 9 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

09156280 BIOSIS NO.: 199497164650
Human endothelial cells treated by oxidized low density lipoproteins express **P-selectin** and bind monocytes.
AUTHOR: McGregor J L; Murphy J; Reck M-P; Gebuhrer V
AUTHOR ADDRESS: INSERM Unit 331, Fac. Med. Alexis Carrel, Pasteur Inst., Lyon**France
JOURNAL: Journal of Cellular Biochemistry Supplement 0 (18 PART A):p271
1994
CONFERENCE/MEETING: Keystone Symposium on Inflammation, Growth Regulatory Molecules and Atherosclerosis Keystone, Colorado, USA January 16-23, 1994

ISSN: 0733-1959
RECORD TYPE: Citation
LANGUAGE: English

16/3/10 (Item 1 from file: 73)
DIALOG(R)File 73:EMBASE
(c) 2001 Elsevier Science B.V. All rts. reserv.

05875679 EMBASE No: 1994288635
Platelet alpha-granule release in cocaine users
Rinder H.M.; Ault K.A.; Jatlow P.I.; Kosten T.R.; Smith B.R.
Department of Laboratory Medicine, Yale University School of Medicine, PO Box 208035,New Haven, CT 06520-8035 United States
Circulation (CIRCULATION) (United States) 1994, 90/3 (1162-1167)
CODEN: CIRCA ISSN: 0009-7322
DOCUMENT TYPE: Journal; Article
LANGUAGE: ENGLISH SUMMARY LANGUAGE: ENGLISH

16/3/11 (Item 1 from file: 399)
DIALOG(R)File 399:CA SEARCH(R)
(c) 2001 AMERICAN CHEMICAL SOCIETY. All rts. reserv.

123310911 CA: 123(23)310911a CONFERENCE PROCEEDING
Endothelium, blood-born cells and intimal colony forming units in human atherogenesis
AUTHOR(S): Balyasnikova, I. V.; Byzova, T. V.; Bystrevskaya, V. B.; Ilyinskaya, O. P.; Krushinsky, A. V.; Popkova, V. M.; Romanov, Yu. A.; Soboleva, E. L.; Tararak, E. M.; Smirnov, V. N.
LOCATION: Cardiology Research Center, Academy Medical Sciences, Moscow, Russia,
JOURNAL: Eur. Sect. Meet., Int. Soc. Heart Res., 15th EDITOR: Haunsoe, Stig (Ed), Kjeldsen, Keld (Ed), DATE: 1994 PAGES: 327-30 CODEN: 61SCA9
LANGUAGE: English PUBLISHER: Monduzzi Editore,Bologna, Italy

16/3/12 (Item 2 from file: 399)
DIALOG(R)File 399:CA SEARCH(R)
(c) 2001 AMERICAN CHEMICAL SOCIETY. All rts. reserv.

121026895 CA: 121(3)26895n PATENT
Cyclic peptide inhibitors of cellular adhesion derived from selectins
INVENTOR(AUTHOR): Heavner, George A.
LOCATION: USA
ASSIGNEE: Centocor, Inc.
PATENT: PCT International ; WO 9405310 A1 DATE: 940317
APPLICATION: WO 93US8504 (930908) *US 941653 (920908)
PAGES: 175 pp. CODEN: PIXXD2 LANGUAGE: English CLASS: A61K-037/00A; A61K-037/02B; C07K-005/00B; C07K-007/00B; C07K-015/00B; C07K-017/00B
DESIGNATED COUNTRIES: CA; JP; US DESIGNATED REGIONAL: AT; BE; CH; DE; DK ; ES; FR; GB; GR; IE; IT; LU; MC; NL; PT; SE

16/3/13 (Item 3 from file: 399)
DIALOG(R)File 399:CA SEARCH(R)
(c) 2001 AMERICAN CHEMICAL SOCIETY. All rts. reserv.

120315824 CA: 120(25)315824x PATENT
P-selectin-derived inhibitors of leukocyte adhesion
INVENTOR(AUTHOR): Heavner, George A.; Epps, Leon A.
LOCATION: USA
ASSIGNEE: Centocor, Inc.
PATENT: PCT International ; WO 9405314 A1 DATE: 940317
APPLICATION: WO 93US7964 (930824) *US 941652 (920908)
PAGES: 56 pp. CODEN: PIXXD2 LANGUAGE: English CLASS: A61K-037/02A;

C07K-007/00B DESIGNATED COUNTRIES: CA; JP DESIGNATED REGIONAL: AT; BE; CH ; DE; DK; ES; FR; GB; GR; IE; IT; LU; MC; NL; PT; SE
? t s16/kwic/all

>>>KWIC option is not available in file(s): 399

16/KWIC/1 (Item 1 from file: 5)
DIALOG(R)File 5:(c) 2001 BIOSIS. All rts. reserv.

1994

ABSTRACT: Platelet activation is associated with acute vessel occlusion and chronic **restenosis** after percutaneous transluminal coronary angioplasty (PTCA). Organic nitrates, which act by releasing the vasodilator and...

...of GSNO. Blood was sampled from the coronary sinus to measure platelet surface expression of **P-selectin** and glycoprotein IIb/IIIa as indices of platelet activation. in 7 control patients, PTCA caused a rise in platelet surface expression of **P-selectin** and glycoprotein IIb/IIIa, which was maximal 5 minutes after PTCA, indicating increased platelet activation...

...min before PTCA. GSNO significantly inhibited the PTCA-induced increase in platelet surface expression of **P-selectin** and glycoprotein IIb/IIIa without altering blood pressure. These findings show that platelets are activated...

16/KWIC/2 (Item 2 from file: 5)
DIALOG(R)File 5:(c) 2001 BIOSIS. All rts. reserv.

Role of **P-selectin** in animal models of thrombosis and **restenosis**.
1994

16/KWIC/3 (Item 3 from file: 5)
DIALOG(R)File 5:(c) 2001 BIOSIS. All rts. reserv.

Increase in the adhesion molecule **P-selectin** in endothelium overlying atherosclerotic plaques: Coexpression with intercellular adhesion molecule-1.
1994

ABSTRACT: **P-selectin** (GMP-140) is an adhesion molecule present within endothelial cells that is rapidly translocated to...

...mediates endothelial-leukocyte interactions. Immunohistochemical analysis of human atherosclerotic plaques has shown strong expression of **P-selectin** by the endothelium overlying active atherosclerotic plaques. **P-selectin** is not, however, detected in normal arterial endothelium or in endothelium overlying inactive fibrous plaques. Color image analysis was used to quantitate the degree of **P-selectin** expression in the endothelium and demonstrates a statistically significant increase in **P-selectin** expression by atherosclerotic endothelial cells. Double immunofluorescence shows that some of this **P-selectin** is expressed on the luminal surface of the endothelial cells. Previous work has demonstrated a...

...in atherosclerotic endothelium and a study on the expression of intercellular adhesion molecule-1 and **P-selectin** in **atherosclerosis** shows a highly positive correlation. These results suggest that the selective and cooperative expression of **P-selectin** and intercellular adhesion molecule-1 may be involved in the recruitment of monocytes into sites of **atherosclerosis**.

16/KWIC/4 (Item 4 from file: 5)
DIALOG(R)File 5:(c) 2001 BIOSIS. All rts. reserv.

1994

...ABSTRACT: novo protein synthesis. Interestingly, E-selectin, intercellular adhesion molecule-1, vascular cell-adhesion molecule or **P-selectin** induction was not apparent in this system suggesting the presence of an alternative system for...
MISCELLANEOUS TERMS: ...**ATHEROSCLEROSIS**; ...

...**P-SELECTIN**;

16/KWIC/5 (Item 5 from file: 5)
DIALOG(R)File 5:(c) 2001 BIOSIS. All rts. reserv.

...cobra venom protease that cleaves the Von Willebrand factor receptor on human platelets and the **P-selectin** receptor on neutrophils.
1994
MISCELLANEOUS TERMS: **ATHEROSCLEROSIS**;

16/KWIC/6 (Item 6 from file: 5)
DIALOG(R)File 5:(c) 2001 BIOSIS. All rts. reserv.

Expression cloning of a functional glycoprotein ligand for **P-selectin**.
1994
MISCELLANEOUS TERMS: ...**ATHEROSCLEROSIS**;

16/KWIC/7 (Item 7 from file: 5)
DIALOG(R)File 5:(c) 2001 BIOSIS. All rts. reserv.

Increase in **P-selectin** in the endothelium overlying human atherosclerotic plaques: Coexpression with ICAM-1.
1994
MISCELLANEOUS TERMS: ...**ATHEROSCLEROSIS**;

16/KWIC/8 (Item 8 from file: 5)
DIALOG(R)File 5:(c) 2001 BIOSIS. All rts. reserv.

Structure/function studies of **P-selectin** glycoprotein ligand.
1994
MISCELLANEOUS TERMS: ...**ATHEROSCLEROSIS**;

16/KWIC/9 (Item 9 from file: 5)
DIALOG(R)File 5:(c) 2001 BIOSIS. All rts. reserv.

Human endothelial cells treated by oxidized low density lipoproteins express **P-selectin** and bind monocytes.
1994
MISCELLANEOUS TERMS: **ATHEROSCLEROSIS**;

16/KWIC/10 (Item 1 from file: 73)
DIALOG(R)File 73:(c) 2001 Elsevier Science B.V. All rts. reserv.

...of circulating activated platelets in whole blood (those that express the alpha-granule membrane protein **P-selectin**), we found that 5 of 25 samples from 12 long-term cocaine users had a...

...at concentrations of 10sup -sup 7 to 10sup -sup 5 mol/L to cause platelet **P-selectin** expression in vitro in this study, coupled with the acute increase in circulating activated platelets...
MEDICAL DESCRIPTORS:
article; **atherosclerosis**--diagnosis--di; clinical article; controlled study; drug dependence--etiology--et; human; human cell; human tissue...
1994
? ds

Set	Items	Description
S1	25	(PSGL? OR P(W)SELECTIN(W)GLYCOPROTEIN(W)LIGAND) AND (ATHEROSCLER?)
S2	14	RD S1 (unique items)
S3	13	(PSGL? OR P(W)SELECTIN(W)GLYCOPROTEIN(W)LIGAND) AND (RESTENOSIS)
S4	9	RD S3 (unique items)
S5	2	S2 AND S4
S6	2	RD S5 (unique items)
S7	3360	RESTENOSIS AND ATHEROSCLEROSIS
S8	495	S7 AND REVIEW?
S9	303	RESTENOSIS (20N) ATHEROSCLEROSIS AND REVIEW?
S10	0	S9 AND P(W)SELECTION?
S11	1	S9 AND P(W)SELECTIN?
S12	511	(RESTENOSIS OR ATHEROSCLEROSIS) AND P(W)SELECTIN?
S13	27	S12 AND REVIEW?
S14	21	RD S13 (unique items)
S15	18	S12 AND PY=1994
S16	13	RD S15 (unique items)

? s s12 and restenosis

	511	S12
	24623	RESTENOSIS
S17	102	S12 AND RESTENOSIS

? s s17 and py=1994

	102	S17
	1920496	PY=1994
S18	4	S17 AND PY=1994

? rd s18

...completed examining records

S19	2	RD S18 (unique items)

? t s19/3/all

19/3/1 (Item 1 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)
(c) 2001 BIOSIS. All rts. reserv.

09627100 BIOSIS NO.: 199598082018
Inhibition of platelet activity by S-nitrosoglutathione during coronary angioplasty.
AUTHOR: Langford E J; Brown A S; Wainwright R J; Debelder A J; Thomas M R; Smith R E A; Radomski M W; Martin J F(a); Moncada S
AUTHOR ADDRESS: (a)Cardiol. Dep., King's Coll. Hosp., London**UK
JOURNAL: Lancet (North American Edition) 344 (8935):p1458-1460 **1994**
ISSN: 0099-5355
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English

19/3/2 (Item 2 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

09566998 BIOSIS NO.: 199598021916
Role of **P-selectin** in animal models of thrombosis and **restenosis**.
AUTHOR: Shebuski Ronald J; Humphrey William R; Simmons Carol A; Hoover Jennifer L; Degraaf Garry L; Geng Jian G; Toombs Christopher F; Anderson Donald C
AUTHOR ADDRESS: Upjohn Lab., Kalamazoo, MI**USA
JOURNAL: Circulation 90 (4 PART 2):pI142 **1994**
CONFERENCE/MEETING: 67th Scientific Sessions of the American Heart Association Dallas, Texas, USA November 14-17, 1994
ISSN: 0009-7322
RECORD TYPE: Citation
LANGUAGE: English

6/7/40 (Item 17 from file: 155)
DIALOG(R)File 155:MEDLINE(R)

09288954 21026284 PMID: 11151785
Cardiac allograft vasculopathy--problem and model.
von Scheidt W
Medizinische Klinik und Poliklinik I Klinikum Grosshadern Ludwig-Maximilians-Universitat Munchen 81366 Munchen, Germany. wolfgang.scheidt@med1.med.uni-muenchen.de
Zeitschrift fur Kardiologie (Germany) 2000, 89 Suppl 9 pIX/2-5,
ISSN 0300-5860 Journal Code: 0360430
Document type: Journal Article
Languages: ENGLISH
Main Citation Owner: NLM
Record type: Completed
Cardiac allograft vasculopathy (CAV) is an accelerated form of atherosclerosis induced by immunological endothelial injury with subsequent inflammatory repair responses in a milieu of additional nonimmunological risk factors. It is the leading cause of death beyond the first year after transplantation. The clinical situation is characterized by a poorly controlled complexity of pathogenetic and protective mechanisms and the heterogeneous nature concerning functional and structural manifestations, disease progression and prognosis. An early risk prediction algorithm for CAV is required in order to establish optimized preventive and therapeutical strategies. Experimental animals serve as model systems to selectively investigate different steps of the injury cascade providing specific insights into key mechanisms operating in CAV. Beyond its importance in transplantation medicine, human CAV can be taken as an unique model of **atherosclerosis** allowing evaluation and **correlation** of vascular function and morphology with the humoral and intracardiac activity/expression of mediators of the disease. Thus, CAV, beyond being a cumbersome clinical problem, represents an unique and attractive model of atherosclerosis in humans offering perspectives beyond the usual.
Record Date Created: 20010109
Record Date Completed: 20010412

6/7/41 (Item 18 from file: 155)
DIALOG(R)File 155:MEDLINE(R)

09263938 20584583 PMID: 11154223
Circulating autoantibodies to oxidized cardiolipin **correlate** with isoprostane F(2alpha)-VI levels and the extent of **atherosclerosis** in ApoE-deficient mice: modulation by vitamin E.
Pratico D; Tangirala R K; Horkko S; Witztum J L; Palinski W; FitzGerald G A
The Center for Experimental Therapeutics, Department of Pharmacology, University of Pennsylvania, Philadelphia, PA 19104, USA. domenico@spirit.gcrc.upenn.edu
Blood (UNITED STATES) Jan 15 2001, 97 (2) p459-64, ISSN 0006-4971 Journal Code: 7603509
Contract/Grant No.: HL57505; HL; NHLBI; M01RR00040; RR; NCRR
Document type: Journal Article
Languages: ENGLISH
Main Citation Owner: NLM
Record type: Completed
Lipid peroxidation plays an important role in atherogenesis. Previous studies suggested that autoantibodies against epitopes of oxidized low-density lipoprotein may indicate the extent or rate of progression of

6/7/29 (Item 6 from file: 155)
DIALOG(R)File 155:MEDLINE(R)
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11481077 98364976 PMID: 9701246

A mouse model of human familial hypercholesterolemia: markedly elevated low density lipoprotein cholesterol levels and severe atherosclerosis on a low-fat chow diet.

Powell-Braxton L; Veniant M; Latvala R D; Hirano K I; Won W B; Ross J; Dybdal N; Zlot C H; Young S G; Davidson N O

Cardiovascular Research, Genentech Inc., South San Francisco, California 94080, USA.

Nature medicine (UNITED STATES) Aug 1998, 4 (8) p934-8, ISSN 1078-8956 Journal Code: 9502015

Contract/Grant No.: DK 42086; DK; NIDDK; HL 18577; HL; NHLBI; HL 38180; HL; NHLBI; +

Comment in Nat Med. 1998 Aug;4(8) 899-900; Comment in PMID 9701240; Erratum in Nat Med 1998 Oct;4(10):1200

Document type: Journal Article

Languages: ENGLISH

Main Citation Owner: NLM

Record type: Completed

Mutations in the low density lipoprotein (LDL) receptor gene cause familial hypercholesterolemia, a human disease characterized by premature atherosclerosis and markedly elevated plasma levels of LDL cholesterol and apolipoprotein (apo) B100. In contrast, mice deficient for the LDL receptor (Ldlr-/-) have only mildly elevated LDL cholesterol levels and little **atherosclerosis** . This difference results from extensive editing of the hepatic apoB mRNA in the mouse, which **limits** apoB100 synthesis in favor of apoB48 synthesis. We have generated Ldlr-/- mice that cannot edit the apoB mRNA and therefore synthesize exclusively apoB100. These mice had markedly elevated LDL cholesterol and apoB100 levels and developed extensive atherosclerosis on a chow diet. This authentic model of human familial hypercholesterolemia will provide a new tool for studying atherosclerosis.

Record Date Created: 19980825

Record Date Completed: 19980825

6/7/30 (Item 7 from file: 155)
DIALOG(R)File 155:MEDLINE(R)
(c) format only 2003 The Dialog Corp. All rts. reserv.

11481071 98364970 PMID: 9701240
State of the art: **atherosclerosis** in a **limited** edition.
Rader D J; FitzGerald G A
Nature medicine (UNITED STATES) Aug 1998, 4 (8) p899-900, ISSN 1078-8956 Journal Code: 9502015
Comment on Nat Med. 1998 Aug;4(8) 934-8; Comment on PMID 9701246
Document type: Comment; News
Languages: ENGLISH
Main Citation Owner: NLM
Record type: Completed
Record Date Created: 19980825
Record Date Completed: 19980825

14006752 BIOSIS NO.: 200300000781
Experimental **atherosclerosis**: A historical overview.
AUTHOR: Moghadasian Mohammed H(a)
AUTHOR ADDRESS: (a)Healthy Heart Program, St. Paul's Hospital, 180-1081 Burrard Street, Vancouver, BC, V6Z 1Y6, Canada**Canada E-Mail: mhmoghad@interchange.ubc.ca
JOURNAL: Life Sciences 70 (8):p855-865 January 11 2002 2002
MEDIUM: print
ISSN: 0024-3205
DOCUMENT TYPE: Article; Literature Review
RECORD TYPE: Abstract
LANGUAGE: English

ABSTRACT: Almost one-hundred years ago the first evidence of experimental **atherosclerosis** was reported. Over the past century, significant advances have been made in the development of animal models of human coronary artery disease. In this minireview, induction of atherosclerotic lesions in several animal models including rodents (mice, rabbits, rats, hamsters, guinea pigs), avian (pigeons, chickens, quail), swine, carnivora (dogs, cats), and non-human primates is discussed. The **limitations** and advantages of the animal models of **atherosclerosis** have been summarized. The transgenic/**knockout** animal models have greatly enhanced our understanding of **atherosclerosis**. Compared to wild-type counterparts, the knockout/transgenic animals develop atherogenesis faster without a need for a highly atherogenic diet. Although almost all investigations support a causal role for increased plasma cholesterol levels in the development of atherosclerotic vascular disease, an increasing body of evidence indicates serious involvement of other factors including oxidative stress, inflammation, infection and other emerging risk factors.
?

9/7/9 (Item 1 from file: 73)
DIALOG(R)File 73:EMBASE

06921616 EMBASE No: 1997206086
Insights into selectin function from **knockout** mice
Frenette P.S.; Wagner D.D.
P.S. Frenette, Center for Blood Research, 800 Huntington Ave., Boston, MA 02114 United States
AUTHOR EMAIL: frenette@cbr.med.harvard.edu
Thrombosis and Haemostasis (THROMB. HAEMOST.) (Germany) 1997, 78/1 (60-64)
CODEN: THHAD ISSN: 0340-6245
DOCUMENT TYPE: Journal; Conference Paper
LANGUAGE: ENGLISH SUMMARY LANGUAGE: ENGLISH
NUMBER OF REFERENCES: 42

The development of animal models through gene targeting was very useful to the selectin field. Selectins are found on endothelium, platelets and leukocytes and, they mediate adhesion among these cell types. The removal of a single selectin gene taught us that **P-selectin** on the vessel wall mediates leukocyte rolling in the absence of inflammation and that all three selectins contribute to leukocyte rolling during inflammation. Similarly, **P-selectin** is responsible for early neutrophil recruitment while the other selectins contribute in later stages. The **knockout** animals also confirmed the important role of L-selectin in lymphocyte homing. Removal of both endothelial selectins uncovered the hidden importance of E-selectin in leukocyte homeostasis and showed that the endothelial selectins were as important for leukocyte extravasation as the leukocyte betainf 2 integrins. The submission of selectin-deficient mice to models of various human diseases can provide invaluable information on conditions in which an anti-selectin therapy may prove beneficial.

9/7/8 (Item 8 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

10163013 BIOSIS NO.: 199698617931
P-selectin knockout: A mouse model for various human diseases.
BOOK TITLE: Ciba Foundation Symposium; Cell adhesion and human disease
AUTHOR: Wagner Denisa D
BOOK AUTHOR/EDITOR: Marsh J; Goode J A: Eds
AUTHOR ADDRESS: Cent. Blood Res., Harvard Med. Sch., 800 Huntington Avenue, Boston, MA 02115**USA
JOURNAL: Ciba Foundation Symposium (198):p2-16 1995
BOOK PUBLISHER: John Wiley and Sons Ltd., Baffin Lane, Chichester PO 19 1UD, England
John Wiley and Sons, Inc., 605 Third Avenue, New York, New York 10158-0012, USA
CONFERENCE/MEETING: Symposium London, England, UK May 17-19, 1994
ISSN: 0300-5208 ISBN: 0-471-95279-6
RECORD TYPE: Citation
LANGUAGE: English

6/7/23 (Item 5 from file: 73)
DIALOG(R)File 73:EMBASE

07625908 EMBASE No: 1999112323
Experimental techniques and models in the study of the development and treatment of abdominal aortic aneurysm
Carrell T.W.G.; Smith A.; Burnand K.G.
T.W.G. Carrell, Academic Department of Surgery, St Thomas' Hospital, London SE1 7EH United Kingdom
British Journal of Surgery (BR. J. SURG.) (United Kingdom) 1999, 86/3 (305-312)
CODEN: BJSUA ISSN: 0007-1323
DOCUMENT TYPE: Journal; Article
LANGUAGE: ENGLISH SUMMARY LANGUAGE: ENGLISH
NUMBER OF REFERENCES: 74

Background: It is still unclear what initiates aneurysmal dilatation and what determines whether or not an aneurysm will continue to expand and rupture. Early detection and operative repair of an abdominal aortic aneurysm (AAA) still remains the only effective means of reducing the high mortality rate associated with the condition. Endovascular techniques are being developed in an attempt to reduce the mortality rate associated with elective repair. A variety of animal models and experimental techniques have been described in the investigation of the pathophysiology of AAA and in the development of improved endovascular surgical and pharmacological therapies. This article discusses these models and techniques, their advantages and some of the problems encountered in extrapolating experimental findings to the human condition. Methods: This review is based on a search of the Medline database from 1966 to March 1998 using recognized key words and text words. A further search was then conducted on references quoted within selected relevant publications. Results and conclusion: Treatment of rodent aortas with intraluminal elastase or periaortic calcium chloride creates reproducible aneurysms that have certain similarities to the human pathology; such aneurysms have been favoured in the investigation of the pathophysiology of aneurysm expansion. However, these models **lack** several of the prominent features of the human lesion, such as **atherosclerosis** and intraluminal thrombosis. The development of gene **knockout** mice may lead to a more analogous aneurysm formation, with associated atherosclerosis. Many large animal models have been used in the development of endovascular techniques but, in general, these do not mimic the human pathophysiology and fail to predict medium- and long-term complications.

6/7/24 (Item 1 from file: 155)
DIALOG(R)File 155:MEDLINE(R)

14812134 22620038 PMID: 12588950
Hypercholesterolemia and changes in lipid and bile acid metabolism in male and female cyp7A1-deficient mice.
Erickson Sandra K; Lear Steven R; Deane Sean; Dubrac Sandrine; Huling Sandra L; Nguyen Lien; Bollineni Jaya S; Shefer Sarah; Hyogo Hideyuki; Cohen David E; Shneider Benjamin; Sehayek Ephraim; Ananthanarayanan Meena; Balasubramaniyan Natarajan; Suchy Fredrick J; Batta Ashok K; Salen Gerald
Department of Medicine, University of California, San Francisco, CA 94143.
Journal of lipid research (United States) 02 16 2003, 44 (5) p1001-9, ISSN 0022-2275 Journal Code: 0376606
Document type: Journal Article
Languages: ENGLISH

Main Citation Owner: NLM
Record type: In Process
Cholesterol 7alpha-hydroxylase, a rate-**limiting** enzyme for bile acid synthesis, has been implicated in genetic susceptibility to **atherosclerosis** . The gene, CYP7A1, encoding a protein with this activity, is expressed normally only in hepatocytes and is highly regulated. Our cyp7A1 gene **knockout** mouse colony, as young adults on a chow diet, is hypercholesterolemic. These mice were characterized extensively to understand how cyp7A1 affects lipid and bile acid homeostasis in different tissue compartments and whether gender plays a modifying role. Both male and female cyp7A1-deficient mice had decreased hepatic LDL receptors, unchanged hepatic cholesterol synthesis, increased intestinal cholesterol synthesis and bile acid transporters, and decreased fecal bile acids but increased fecal sterols. In females, cyp7A1 deficiency also caused changes in hepatic fatty acid metabolism, decreased hepatic canalicular bile acid transporter, Bsep, and gallbladder bile composition altered to a lithogenic profile. Taken together, the data suggest that cyp7A1 deficiency results in a proatherogenic phenotype in both genders and leads to a prolithogenic phenotype in females.
Record Date Created: 20030507

6/7/25 (Item 2 from file: 155)
DIALOG(R)File 155:MEDLINE(R)

14497882 22466635 PMID: 12578865
Reciprocal association of C-reactive protein with adiponectin in blood stream and adipose tissue.
Ouchi Noriyuki; Kihara Shinji; Funahashi Tohru; Nakamura Tadashi; Nishida Makoto; Kumada Masahiro; Okamoto Yoshihisa; Ohashi Koji; Nagaretani Hiroyuki; Kishida Ken; Nishizawa Hitoshi; Maeda Norikazu; Kobayashi Hideki; Hiraoka Hisatoyo; Matsuzawa Yuji
Department of Internal Medicine and Molecular Science, Graduate School of Medicine, Osaka University, Suita, Osaka, Japan. ouchi@imed2.med.osaka-u.ac.jp
Circulation (United States) Feb 11 2003, 107 (5) p671-4, ISSN 1524-4539 Journal Code: 0147763
Document type: Clinical Trial; Controlled Clinical Trial; Journal Article
Languages: ENGLISH
Main Citation Owner: NLM

9/7/16 (Item 7 from file: 155)
DIALOG(R)File 155:MEDLINE(R)

09468946 21242601 PMID: 11344083

Direct viewing of **atherosclerosis** in vivo: plaque invasion by leukocytes is initiated by the endothelial selectins.

Eriksson E E; Xie X; Werr J; Thoren P; Lindbom L
Department of Physiology and Pharmacology, Karolinska Institutet, Stockholm, Sweden. einar.eriksson@fyfa.ki.se

FASEB journal - official publication of the Federation of American Societies for Experimental Biology (United States) May 2001, 15 (7) p1149-57, ISSN 0892-6638 Journal Code: 8804484
Document type: Journal Article
Languages: ENGLISH
Main Citation Owner: NLM
Record type: Completed

Leukocyte infiltration in **atherosclerosis** has been extensively investigated by using histological techniques on fixed tissues. In this study, intravital microscopic observations of leukocyte recruitment in the aorta of atherosclerotic mice were performed. Interactions between leukocytes and atherosclerotic endothelium were highly transient, thereby limiting the ability for rolling leukocytes to firmly adhere. Leukocyte rolling was abolished by function inhibition of **P-selectin** (P<0.001, n=8), whereas antibody blockage of E-selectin (n=10) decreased rolling leukocyte flux to 51 +/- 9.9% (mean+/-SE, P<0.01) and increased leukocyte rolling velocity to 162 +/- 18% (P<0.01) of pretreatment values. Notably, function inhibition of the integrin alpha(4) subunit (n=5) had no effect on rolling flux (107+/-25%, P=0.782) or rolling velocity (89+/-6.1%, P=0.147), despite endothelial expression of vascular cell adhesion molecule 1 (VCAM-1). Leukocytes interacting with atherosclerotic endothelium were predominantly neutrophils, because treatment with antineutrophil serum decreased rolling and neutrophil counts in peripheral blood to the same extent. In conclusion, we present the first direct observations of **atherosclerosis** in vivo. We show that transient dynamics of leukocyte-endothelium interactions are important regulators of arterial leukocyte recruitment and that leukocyte rolling in **atherosclerosis** is critically dependent on the endothelial selectins. This experimental technique and the data presented introduce a novel perspective for the study of pathophysiological events involved in large-vessel disease.

/12 (Item 3 from file: 155)
DIALOG(R)File 155:MEDLINE(R)

14599823 22402159 PMID: 12483207

Circulating activated platelets exacerbate **atherosclerosis** in mice deficient in apolipoprotein E.

Huo Yuqing; Schober Andreas; Forlow S Bradley; Smith David F; Hyman Matthew Craig; Jung Steffen; Littman Dan R; Weber Christian; Ley Klaus

Department of Biomedical Engineering and Cardiovascular Research Center, University of Virginia, Health Science Center, Charlottesville, Virginia, USA.

Nature medicine (United States) Jan 2003, 9 (1) p61-7, ISSN 1078-8956 Journal Code: 9502015

Contract/Grant No.: HL-58108; HL; NHLBI

Document type: Journal Article

Languages: ENGLISH

Main Citation Owner: NLM

Record type: Completed

We studied whether circulating activated platelets and platelet-leukocyte aggregates cause the development of atherosclerotic lesions in apolipoprotein-E-deficient (Apoe(-/-)) mice. Circulating activated platelets bound to leukocytes, preferentially monocytes, to form platelet-monocyte/leukocyte aggregates. Activated platelets and platelet-leukocyte aggregates interacted with atherosclerotic lesions. The interactions of activated platelets with monocytes and atherosclerotic arteries led to delivery of the platelet-derived chemokines CCL5 (regulated on activation, normal T cell expressed and secreted, RANTES) and CXCL4 (platelet factor 4) to the monocyte surface and endothelium of atherosclerotic arteries. The presence of activated platelets promoted leukocyte binding of vascular cell adhesion molecule-1 (VCAM-1) and increased their adhesiveness to inflamed or atherosclerotic endothelium. Injection of activated wild-type, but not **P-selectin**-deficient, platelets increased monocyte arrest on the surface of atherosclerotic lesions and the size of atherosclerotic lesions in Apoe(-/-) mice. Our results indicate that circulating activated platelets and platelet-leukocyte/monocyte aggregates promote formation of atherosclerotic lesions. This role of activated platelets in **atherosclerosis** is attributed to platelet **P-selectin** -mediated delivery of platelet-derived proinflammatory factors to monocytes/leukocytes and the vessel wall.

Record Date Created: 20030106

9/7/11 (Item 2 from file: 155)
DIALOG(R)File 155:MEDLINE(R)
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14864857 22617820 PMID: 12707243
Single injection of **P-selectin** or **P-selectin** glycoprotein ligand-1 monoclonal antibody blocks neointima formation after arterial injury in apolipoprotein E-deficient mice.

Phillips J William; Barringhaus Kurt G; Sanders John M; Hesselbacher Sean E; Czarnik Ann C; Manka David; Vestweber Dietmar; Ley Klaus; Sarembock Ian J

Department of Medicine, Cardiovascular Division, University of Virginia Health System, Box 800158, Charlottesville, VA 22908-0158, USA.

Circulation (United States) 04 21 2003, 107 (17) p2244-9, ISSN 1524-4539 Journal Code: 0147763

Contract/Grant No.: HL-58108; HL; NHLBI; HL-66264; HL; NHLBI; T32-HL-07355; HL; NHLBI

Comment in Circulation. 2003 May 6;107(17) 2175-7; Comment in PMID 12732592

Document type: Journal Article

Languages: ENGLISH

Main Citation Owner: NLM

Record type: Completed

BACKGROUND: Emerging data suggest that **P-selectin**, by controlling adhesion of white blood cells, may be important in limiting the response to vascular injury. METHODS AND RESULTS: We tested the hypothesis that transient inhibition of **P-selectin** with either anti-**P-selectin** monoclonal antibody (mAb) or anti-**P-selectin** glycoprotein ligand-1 (PSGL-1) mAb would reduce neointima formation in the setting of carotid denudation injury in **atherosclerosis**-prone apolipoprotein E-/- mice. Neointima formation at 28 days was reduced significantly, by 50% or 80%, by a single injection on the day of injury of 100 or 200 microg **P-selectin** mAb RB 40.34 and by 55% by a single injection of 100 microg PSGL-1 mAb 4RA10 (P< or =0.005). In addition, there was a significant reduction in neointimal macrophage content. CONCLUSIONS: These findings demonstrate that transient **P-selectin** or PSGL-1 blockade at the time of arterial injury significantly limits plaque macrophage content and neointima formation in a dose-dependent manner after carotid denudation injury in apolipoprotein E-/- mice.

Record Date Created: 20030506

Record Date Completed: 20030515

10 (Item 1 from file: 155)
DIALOG(R)File 155:MEDLINE(R)

14883077 22528551 PMID: 12480714
Platelet **P-selectin** facilitates atherosclerotic lesion development.
Burger Peter C; Wagner Denisa D
Center for Blood Research and Department of Pathology, Harvard Medical School, Boston, MA 02115, USA.
Blood (United States) 12 12 2002, 101 (7) p2661-6, ISSN 0006-4971 Journal Code: 7603509
Contract/Grant No.: R01 HL 53756; HL; NHLBI
Document type: Journal Article
Languages: ENGLISH
Main Citation Owner: NLM
Record type: Completed
P-selectin is an adhesion molecule expressed on activated platelets and endothelium. It is known to play an important role in **atherosclerosis**. **P-selectin** also circulates in plasma in a soluble form (sP-selectin), which induces procoagulant microparticle formation. We investigated the role of platelet versus endothelial **P-selectin** in generating sP-selectin and in the formation of atherosclerotic lesions in the apolipoprotein E (apoE)-deficient mouse model. For this we transplanted apoE(-/-)**P-selectin**(-/-) and apoE(-/-)**P-selectin** (+/+) lethally irradiated mice with bone marrow of either genotype. Seven months after transplantation, we determined from the chimeric animals that the majority of circulating sP-selectin was of endothelial origin. Thus, in **atherosclerosis**, the procoagulant sP-selectin reflects endothelial rather than platelet activation. We found that endothelial **P-selectin** was crucial for the promotion of atherosclerotic lesion growth because in its absence only relatively small lesions developed. However, platelet **P-selectin** also contributed to the lesion development because lesions in wild-type recipients receiving transplants with wild-type platelets were 30% larger than those receiving **P-selectin**-deficient platelets (P <.008) and were more frequently calcified (80% versus 44%). In comparison with **P-selectin** wild-type animals, absence of either endothelial or platelet **P-selectin** inhibited migration of smooth muscle cells into the lesion. Thus, in addition to endothelium, platelets and their **P-selectin** also actively promote advanced atherosclerotic lesion development.
Record Date Created: 20030318

knockout and (atherosclerosis) and p(w)selectin
53379 KNOCKOUT
158802 ATHEROSCLEROSIS
4181235 P
30362 SELECTIN
11513 P(W)SELECTIN
S8 19 KNOCKOUT AND (ATHEROSCLEROSIS) AND P(W)SELECTIN
? rd s18
>>>Set 18 has not yet been created.
? rd s8
...completed examining records
S9 16 RD S8 (unique items)
? t s9/7/all

9/7/1 (Item 1 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

14085730 BIOSIS NO.: 200300079759
Vascular and platelet expression of plasminogen activator inhibitor-1 contributes to the prothrombotic phenotype of apoE-**knockout** mice.
AUTHOR: Schaefer Katrin(a); Hecke Anneke(a); Mueller Katja(a); Goebel Julia (a); Mounier Emmanuelle(a); Konstantinides Stavros(a)
AUTHOR ADDRESS: (a)Univ of Goettingen, Goettingen, Germany**Germany
JOURNAL: Circulation 106 (19 Supplement):pII-80 November 5 2002 2002
MEDIUM: print
CONFERENCE/MEETING: Abstracts from Scientific Sessions Chicago, IL, USA November 17-20, 2002
SPONSOR: American Heart Association
ISSN: 0009-7322
RECORD TYPE: Citation
LANGUAGE: English

9/7/2 (Item 2 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

13889124 BIOSIS NO.: 200200517945
Deposition of platelet RANTES triggering monocyte recruitment requires **P-selectin** and is involved in neointima formation after arterial injury.
AUTHOR: Schober Andreas; Manka David; von Hundelshausen Philipp; Huo Yuqing ; Hanrath Peter; Sarembock Ian J; Ley Klaus; Weber Christian(a)
AUTHOR ADDRESS: (a)Kardiovaskulaere Molekularbiologie, Universitaetsklinikum Aachen, Pauwelsstrasse 30, 52074, Aachen**Germany
E-Mail: cweber@ukaachen.de
JOURNAL: Circulation 106 (12):p1523-1529 September 17, 2002
MEDIUM: print
ISSN: 0009-7322
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English

ABSTRACT: Background: Chemokines expressed on atherosclerotic endothelium or deposited by activated platelets have been implicated in monocyte recruitment during atherogenesis and restenosis. Although the involvement of **P-selectin** in these processes is evident from studies in **knockout** mice, it has not been elucidated whether delivery of platelet chemokines requires **P-selectin**, thus serving as a **P-selectin**-dependent effector function. Methods and Results: Using immunofluorescence and laminar flow assays, we found that the deposition of the platelet-derived chemokine RANTES and monocyte arrest subsequently triggered by RANTES immobilized on inflamed endothelium are

more efficient after preperfusion than after static preincubation of platelets and appear to depend on interactions of platelet but not endothelial **P-selectin**. This was revealed by the effects of **P-selectin** antibodies and comparison of **P-selectin**-deficient and wild-type platelets. Immunohistochemistry detected a substantial luminal expression of RANTES on neointimal lesions in wire-injured carotid arteries of apolipoprotein E (apoE)-deficient mice but not of mice with a combined deficiency in apoE and **P-selectin** (or platelet **P-selectin**). As assessed by histomorphometry, treatment of apoE-deficient mice with the RANTES receptor antagonist Met-RANTES markedly reduced neointimal plaque area and macrophage infiltration. Conclusions: Our data suggest that RANTES deposition and subsequent monocyte arrest are promoted by platelet **P-selectin** and involved in wire-induced intimal hyperplasia, and that blocking RANTES receptors attenuates neointima formation and macrophage infiltration. This mechanism represents an important component explaining the protection against neointimal growth in **P-selectin**-deficient mice and may represent a novel approach to the treatment of restenosis or **atherosclerosis** by the administration of chemokine receptor antagonists.

9/7/3 (Item 3 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

13646022 BIOSIS NO.: 200200274843
A high-cholesterol diet leads to vascular inflammation and **atherosclerosis** in SR-BI deficient mice.
AUTHOR: Twisk Jaap(a); Van Eck Miranda(a); Bos Sophie(a); van Berkel Theo J C(a)
AUTHOR ADDRESS: (a)Leiden University/LACDR, Leiden**Netherlands
JOURNAL: Circulation 104 (17 Supplement):pII241-II242 October 23, 2001
MEDIUM: print
CONFERENCE/MEETING: Scientific Sessions 2001 of the American Heart Association Anaheim, California, USA November 11-14, 2001
ISSN: 0009-7322
RECORD TYPE: Citation
LANGUAGE: English

9/7/4 (Item 4 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

13634402 BIOSIS NO.: 200200263223
Peptide antagonists to **p-selectin**: Potential in anti-atherothrombotic therapy.
AUTHOR: Molenaar Tom J M(a); Twisk Jaap(a); Appeldoorn Chantal A M(a); de Haas Sonja A M(a); Michon Ingrid(a); van Berkel Theo J C(a); Kuiper Johan ; Biessen Erik A L
AUTHOR ADDRESS: (a)Leiden Univ, Leiden**Netherlands
JOURNAL: Circulation 104 (17 Supplement):pII38-II39 October 23, 2001
MEDIUM: print
CONFERENCE/MEETING: Scientific Sessions 2001 of the American Heart Association Anaheim, California, USA November 11-14, 2001
ISSN: 0009-7322
RECORD TYPE: Citation
LANGUAGE: English

9/7/5 (Item 5 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

13329489 BIOSIS NO.: 200100536638
Adhesion molecules and atherogenesis.
AUTHOR: Huo Y; Ley K(a)
AUTHOR ADDRESS: (a)Department of Biomedical Engineering, Health Science Center, University of Virginia, Charlottesville, VA, 22908**USA
JOURNAL: Acta Physiologica Scandinavica 173 (1):p35-43 September, 2001
MEDIUM: print
ISSN: 0001-6772
DOCUMENT TYPE: Literature Review
RECORD TYPE: Abstract
LANGUAGE: English
SUMMARY LANGUAGE: English

ABSTRACT: **Atherosclerosis** is an inflammatory disease of the vessel wall characterized by monocyte infiltration in response to pro-atherogenic factors such as oxidized lipids. Recently, the role of specific adhesion molecules in this process has been explored. The endothelium overlying atherosclerotic lesions expresses **P-selectin** and the shoulder regions express vascular cell adhesion molecule-1 (VCAM-1) and intercellular adhesion molecule-1 (ICAM-1), which is also expressed on endothelium in regions not prone to plaque development. Serum levels of soluble **P-selectin**, ICAM-1 and VCAM-1 are elevated in patients with angina pectoris or peripheral atherosclerotic disease. Reconstituted in vitro systems using monocytes on cytokine-activated endothelial cells under shear flow suggested the involvement of **P-selectin**, L-selectin, VCAM-1, its ligand, VLA-4 integrin and CD18 integrins. Studies of monocyte adhesion in isolated perfused carotid arteries harvested from atherosclerotic (apoE-/-) mice show a predominant involvement of **P-selectin** and its ligand **P-selectin** glycoprotein-1 (PSGL-1) in rolling and of VLA-4 and VCAM-1 in firm adhesion. Consistent with these findings, apoE-/- mice that are also deficient for **P-selectin** show significantly reduced atherosclerotic lesion sizes and are almost completely protected from neointimal growth after vascular injury. Milder effects are also seen in the low-density lipoprotein (LDL) receptor deficient (LDLR-/-) mouse. In a high cholesterol/cholate model, a role of ICAM-1 and CD18 integrins was also shown, but this awaits confirmation in more physiologic models. Transient blockade of the VLA-4/VCAM-1 adhesion pathway by antibodies or peptides in apoE-/- or LDLR-/- mice reduced monocyte and lipid accumulation in lesions. These data suggest that **P-selectin**, PSGL-1, VLA-4 and VCAM-1 are the most important adhesion molecules involved in monocyte recruitment to atherosclerotic lesions.

9/7/6 (Item 6 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

12904893 BIOSIS NO.: 200100112042
Expression of SR-B1 in atherosclerotic lesions from apoE-deficient mice is inversely correlated with the severity of lesion development.
AUTHOR: Twisk Jaap(a); Van Berkel Theo J C(a)
AUTHOR ADDRESS: (a)Leiden/Amsterdam Ctr for Drug Research, Leiden** Netherlands
JOURNAL: Circulation 102 (18 Supplement):pII48 October 31, 2000
MEDIUM: print
CONFERENCE/MEETING: Abstracts from Scientific Sessions 2000 New Orleans, Louisiana, USA November 12-15, 2000
ISSN: 0009-7322
RECORD TYPE: Citation
LANGUAGE: English
SUMMARY LANGUAGE: English

9/7/7 (Item 7 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)
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11909303 BIOSIS NO.: 199900155412
Donor and recipient contributions of ICAM-1 and **P-selectin** in parenchymal rejection and graft arteriosclerosis: Insights from double **knockout** mice.
AUTHOR: Raisanen-Sokolowski A K; Glysing-Jensen T; Russell M E
AUTHOR ADDRESS: Cardiovascular Biol. Lab., Harvard Sch. Public Health, Brigham and Women's Hosp., Harvard Med. Sch.,**USA
JOURNAL: Journal of Heart and Lung Transplantation 18 (1):p61 Jan., 1999
CONFERENCE/MEETING: Nineteenth Annual Meeting and Scientific Sessions of the International Society for Heart and Lung Transplantation San Francisco, California, USA April 21-24, 1999
SPONSOR: International Society for Heart and Lung Transplantation
ISSN: 1053-2498
RECORD TYPE: Citation
LANGUAGE: English

9/7/8 (Item 8 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)
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10163013 BIOSIS NO.: 199698617931
P-selectin knockout: A mouse model for various human diseases.
BOOK TITLE: Ciba Foundation Symposium; Cell adhesion and human disease
AUTHOR: Wagner Denisa D
BOOK AUTHOR/EDITOR: Marsh J; Goode J A: Eds
AUTHOR ADDRESS: Cent. Blood Res., Harvard Med. Sch., 800 Huntington Avenue, Boston, MA 02115**USA
JOURNAL: Ciba Foundation Symposium (198):p2-16 1995
BOOK PUBLISHER: John Wiley and Sons Ltd., Baffin Lane, Chichester PO 19 1UD, England
John Wiley and Sons, Inc., 605 Third Avenue, New York, New York 10158-0012, USA
CONFERENCE/MEETING: Symposium London, England, UK May 17-19, 1994
ISSN: 0300-5208 ISBN: 0-471-95279-6
RECORD TYPE: Citation
LANGUAGE: English

9/7/9 (Item 1 from file: 73)
DIALOG(R)File 73:EMBASE
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06921616 EMBASE No: 1997206086
Insights into selectin function from **knockout** mice
Frenette P.S.; Wagner D.D.
P.S. Frenette, Center for Blood Research, 800 Huntington Ave., Boston, MA 02114 United States
AUTHOR EMAIL: frenette@cbr.med.harvard.edu
Thrombosis and Haemostasis (THROMB. HAEMOST.) (Germany) 1997, 78/1 (60-64)
CODEN: THHAD ISSN: 0340-6245
DOCUMENT TYPE: Journal; Conference Paper
LANGUAGE: ENGLISH SUMMARY LANGUAGE: ENGLISH
NUMBER OF REFERENCES: 42

The development of animal models through gene targeting was very useful

to the selectin field. Selectins are found on endothelium, platelets and leukocytes and, they mediate adhesion among these cell types. The removal of a single selectin gene taught us that **P-selectin** on the vessel wall mediates leukocyte rolling in the absence of inflammation and that all three selectins contribute to leukocyte rolling during inflammation. Similarly, **P-selectin** is responsible for early neutrophil recruitment while the other selectins contribute in later stages. The **knockout** animals also confirmed the important role of L-selectin in lymphocyte homing. Removal of both endothelial selectins uncovered the hidden importance of E-selectin in leukocyte homeostasis and showed that the endothelial selectins were as important for leukocyte extravasation as the leukocyte betainf 2 integrins. The submission of selectin-deficient mice to models of various human diseases can provide invaluable information on conditions in which an anti-selectin therapy may prove beneficial.

9/7/10 (Item 1 from file: 155)
DIALOG(R)File 155:MEDLINE(R)

14883077 22528551 PMID: 12480714
Platelet **P-selectin** facilitates atherosclerotic lesion development.
Burger Peter C; Wagner Denisa D
Center for Blood Research and Department of Pathology, Harvard Medical School, Boston, MA 02115, USA.
Blood (United States) 12 12 2002, 101 (7) p2661-6, ISSN 0006-4971 Journal Code: 7603509
Contract/Grant No.: R01 HL 53756; HL; NHLBI
Document type: Journal Article
Languages: ENGLISH
Main Citation Owner: NLM
Record type: Completed
P-selectin is an adhesion molecule expressed on activated platelets and endothelium. It is known to play an important role in **atherosclerosis**. **P-selectin** also circulates in plasma in a soluble form (sP-selectin), which induces procoagulant microparticle formation. We investigated the role of platelet versus endothelial **P-selectin** in generating sP-selectin and in the formation of atherosclerotic lesions in the apolipoprotein E (apoE)-deficient mouse model. For this we transplanted apoE(-/-)**P-selectin**(-/-) and apoE(-/-)**P-selectin** (+/+) lethally irradiated mice with bone marrow of either genotype. Seven months after transplantation, we determined from the chimeric animals that the majority of circulating sP-selectin was of endothelial origin. Thus, in **atherosclerosis**, the procoagulant sP-selectin reflects endothelial rather than platelet activation. We found that endothelial **P-selectin** was crucial for the promotion of atherosclerotic lesion growth because in its absence only relatively small lesions developed. However, platelet **P-selectin** also contributed to the lesion development because lesions in wild-type recipients receiving transplants with wild-type platelets were 30% larger than those receiving **P-selectin**-deficient platelets (P <.008) and were more frequently calcified (80% versus 44%). In comparison with **P-selectin** wild-type animals, absence of either endothelial or platelet **P-selectin** inhibited migration of smooth muscle cells into the lesion. Thus, in addition to endothelium, platelets and their **P-selectin** also actively promote advanced atherosclerotic lesion development.
Record Date Created: 20030318
Record Date Completed: 20030521

9/7/11 (Item 2 from file: 155)

DIALOG(R)File 155:MEDLINE(R)
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14864857 22617820 PMID: 12707243
Single injection of **P-selectin** or **P-selectin** glycoprotein ligand-1 monoclonal antibody blocks neointima formation after arterial injury in apolipoprotein E-deficient mice.
Phillips J William; Barringhaus Kurt G; Sanders John M; Hesselbacher Sean E; Czarnik Ann C; Manka David; Vestweber Dietmar; Ley Klaus; Sarembock Ian J
Department of Medicine, Cardiovascular Division, University of Virginia Health System, Box 800158, Charlottesville, VA 22908-0158, USA.
Circulation (United States) 04 21 2003, 107 (17) p2244-9, ISSN 1524-4539 Journal Code: 0147763
Contract/Grant No.: HL-58108; HL; NHLBI; HL-66264; HL; NHLBI; T32-HL-07355; HL; NHLBI
Comment in Circulation. 2003 May 6;107(17) 2175-7; Comment in PMID 12732592
Document type: Journal Article
Languages: ENGLISH
Main Citation Owner: NLM
Record type: Completed
BACKGROUND: Emerging data suggest that **P-selectin** , by controlling adhesion of white blood cells, may be important in limiting the response to vascular injury. METHODS AND RESULTS: We tested the hypothesis that transient inhibition of **P-selectin** with either anti-**P-selectin** monoclonal antibody (mAb) or anti-**P-selectin** glycoprotein ligand-1 (PSGL-1) mAb would reduce neointima formation in the setting of carotid denudation injury in **atherosclerosis** -prone apolipoprotein E-/- mice. Neointima formation at 28 days was reduced significantly, by 50% or 80%, by a single injection on the day of injury of 100 or 200 microg **P-selectin** mAb RB 40.34 and by 55% by a single injection of 100 microg PSGL-1 mAb 4RA10 (P< or =0.005). In addition, there was a significant reduction in neointimal macrophage content. CONCLUSIONS: These findings demonstrate that transient **P-selectin** or PSGL-1 blockade at the time of arterial injury significantly limits plaque macrophage content and neointima formation in a dose-dependent manner after carotid denudation injury in apolipoprotein E-/- mice.
Record Date Created: 20030506
Record Date Completed: 20030515

9/7/12 (Item 3 from file: 155)
DIALOG(R)File 155:MEDLINE(R)
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14599823 22402159 PMID: 12483207
Circulating activated platelets exacerbate **atherosclerosis** in mice deficient in apolipoprotein E.
Huo Yuqing; Schober Andreas; Forlow S Bradley; Smith David F; Hyman Matthew Craig; Jung Steffen; Littman Dan R; Weber Christian; Ley Klaus
Department of Biomedical Engineering and Cardiovascular Research Center, University of Virginia, Health Science Center, Charlottesville, Virginia, USA.
Nature medicine (United States) Jan 2003, 9 (1) p61-7, ISSN 1078-8956 Journal Code: 9502015
Contract/Grant No.: HL-58108; HL; NHLBI
Document type: Journal Article
Languages: ENGLISH
Main Citation Owner: NLM
Record type: Completed
We studied whether circulating activated platelets and platelet-leukocyte aggregates cause the development of atherosclerotic lesions in

apolipoprotein-E-deficient (Apoe(-/-)) mice. Circulating activated platelets bound to leukocytes, preferentially monocytes, to form platelet-monocyte/leukocyte aggregates. Activated platelets and platelet-leukocyte aggregates interacted with atherosclerotic lesions. The interactions of activated platelets with monocytes and atherosclerotic arteries led to delivery of the platelet-derived chemokines CCL5 (regulated on activation, normal T cell expressed and secreted, RANTES) and CXCL4 (platelet factor 4) to the monocyte surface and endothelium of atherosclerotic arteries. The presence of activated platelets promoted leukocyte binding of vascular cell adhesion molecule-1 (VCAM-1) and increased their adhesiveness to inflamed or atherosclerotic endothelium. Injection of activated wild-type, but not **P-selectin**-deficient, platelets increased monocyte arrest on the surface of atherosclerotic lesions and the size of atherosclerotic lesions in Apoe(-/-) mice. Our results indicate that circulating activated platelets and platelet-leukocyte/monocyte aggregates promote formation of atherosclerotic lesions. This role of activated platelets in **atherosclerosis** is attributed to platelet **P-selectin** -mediated delivery of platelet-derived proinflammatory factors to monocytes/leukocytes and the vessel wall.

Record Date Created: 20030106
Record Date Completed: 20030318

9/7/13 (Item 4 from file: 155)
DIALOG(R)File 155:MEDLINE(R)

10095398 22052265 PMID: 12057918

Atherosclerotic lesions grow through recruitment and proliferation of circulating monocytes in a murine model.

Lessner Susan M; Prado Heather L; Waller Edmund K; Galis Zorina S
Division of Cardiology, Emory University School of Medicine, Atlanta, Georgia 30322, USA.

American journal of pathology (United States) Jun 2002, 160 (6) p2145-55, ISSN 0002-9440 Journal Code: 0370502
Contract/Grant No.: 5 T32 HL07745 07; HL; NHLBI
Document type: Journal Article
Languages: ENGLISH
Main Citation Owner: NLM
Record type: Completed

Macrophage-derived foam cells in developing atherosclerotic lesions may potentially originate either from recruitment of circulating monocytes or from migration of resident tissue macrophages. In this study, we have determined the source of intimal macrophages in the apoE-**knockout** mouse flow-cessation/hypercholesterolemia model of **atherosclerosis** using a bone marrow transplantation approach. We also examined the time course and spatial distribution of intercellular adhesion molecule-1 and vascular cell adhesion molecule-1 expression to assess whether endothelial adhesion molecules were involved in recruitment of either circulating monocytes or resident macrophages. We used allelic variants of the mouse common leukocyte antigen (CD45) to distinguish host-derived and donor-derived white blood cells (WBCs) both in blood and in macrophage-rich carotid lesions. We found that the distribution of CD45 isoforms in lesions is similar to that of circulating WBCs, whereas the host-type CD45 isoform is more prevalent in resident adventitial macrophages. These data indicate that macrophage-derived foam cells in the lesion derive mainly from circulating precursors rather than from resident macrophages. The corresponding time course of intercellular adhesion molecule-1 and vascular cell adhesion molecule-1 expression suggests that recruitment of circulating WBCs by endothelial adhesion molecules is likely to be more important during lesion initiation than during the later phase of rapid lesion growth.

Record Date Created: 20020611

Record Date Completed: 20020715

9/7/14 (Item 5 from file: 155)
DIALOG(R)File 155:MEDLINE(R)

09689155 21481241 PMID: 11597929
S17834, a new inhibitor of cell adhesion and **atherosclerosis** that targets nadph oxidase.
Cayatte A J; Rupin A; Oliver-Krasinski J; Maitland K; Sansilvestri-Morel P; Boussard M F; Wierzbicki M; Verbeuren T J; Cohen R A
Vascular Biology Unit, Boston University Medical Center, Boston, Massachusetts, USA.
Arteriosclerosis, thrombosis, and vascular biology (United States) Oct 2001, 21 (10) p1577-84, ISSN 1524-4636 Journal Code: 9505803
Contract/Grant No.: HL-31607; HL; NHLBI; HL-51875; HL; NHLBI; HL-55620; HL; NHLBI
Document type: Journal Article
Languages: ENGLISH
Main Citation Owner: NLM
Record type: Completed
microdant stress is involved in the events that accompany endothelial cell expression of adhesion molecules and leukocyte adherence in many disease states, including **atherosclerosis** . A recently discovered benzo(b)pyran-4-one derivative, S17834 (10 to 50 micromol/L), reduced tumor necrosis factor-stimulated vascular cell adhesion molecule-1 (VCAM) mRNA accumulation and protein expression in human umbilical vein endothelial cells. Intercellular cell adhesion molecule-1 and E-selectin were also inhibited by S17834, but platelet endothelial cell adhesion molecule-1 was not. Adherence of U937 monocytic cells to the endothelial cells as well as to plastic plates coated with soluble VCAM, intercellular cell adhesion molecule-1, **P-selectin** , and E-selectin was also decreased.
Consistent with an antioxidant mechanism of action, S17834 (10 to 50 micromol/L) inhibited tumor necrosis factor-stimulated release of superoxide from endothelial cells measured by cytochrome c reduction. S17834 had no effect on superoxide produced by xanthine oxidase, indicating that rather than by acting as a scavenger of superoxide anion, the drug acts by inhibiting the production of free radicals. Indeed, S17834 inhibited NADPH oxidase activity of endothelial cell membranes. The ability to inhibit superoxide anion production appears to be key in the effect of S17834 on superoxide anion production and VCAM expression, because these actions were mimicked by adenovirus-mediated overexpression of superoxide dismutase. Furthermore, these actions may be relevant in vivo, because S17834 reduced aortic superoxide anion levels by 40% and aortic atherosclerotic lesions by 60% in apolipoprotein E-deficient mice. These results indicate that S17834 inhibits adhesion molecule expression and adherence of leukocytes to endothelial cells as well as aortic atherogenesis and that perhaps these effects can be explained by its ability to inhibit endogenous superoxide anion production.
Record Date Created: 20011012
Record Date Completed: 20011204

9/7/15 (Item 6 from file: 155)
DIALOG(R)File 155:MEDLINE(R)

09625372 21411458 PMID: 11520791
Localized reduction of **atherosclerosis** in von Willebrand factor-deficient mice.
Methia N; Andre P; Denis C V; Economopoulos M; Wagner D D
Center for Blood Research and the Department of Pathology, Harvard Medical School, Boston, MA 02115, USA.

Blood (United States) Sep 1 2001, 98 (5) p1424-8, ISSN 0006-4971
Journal Code: 7603509
Contract/Grant No.: R37 HL41002; HL; NHLBI
Document type: Journal Article
Languages: ENGLISH
Main Citation Owner: NLM
Record type: Completed

To examine the role of the platelet adhesion molecule von Willebrand factor (vWf) in atherogenesis, vWf-deficient mice (vWf-/-) were bred with mice lacking the low-density lipoprotein receptor (LDLR-/-) on a C57BL/6J background. LDLR-/-vWf+/+ and LDLR-/-vWf-/- mice were placed on a diet rich in saturated fat and cholesterol for different lengths of time. The atherogenic diet stimulated leukocyte rolling in the mesenteric venules in both genotypes, indicating an increase in **P-selectin**-mediated adhesion to the endothelium. After 8 weeks on the atherogenic diet, the fatty streaks formed in the aortic sinus of LDLR-/-vWf-/- mice of either sex were 40% smaller and contained fewer monocytes than those in LDLR-/-vWf+/+ mice. After 22 weeks on the atherogenic diet (early fibrous plaque stage), the difference in lesion size in the aortic sinus persisted. Interestingly, the lesion distribution in the aortas of LDLR-/-vWf-/- animals was different from that of LDLR-/- vWf+/+ animals. In vWf-positive mice, half of all lesions were located at the branch points of the renal and mesenteric arteries, whereas lesions in this area were not as prominent in the vWf-negative mice. These results indicate that the absence of vWf primarily affects the regions of the aorta with disturbed flow that are prone to **atherosclerosis**. Thus, vWf may recruit platelets/leukocytes to the lesion in a flow-dependent manner or may be part of the mechano-transduction pathway regulating endothelial response to shear stress.

Record Date Created: 20010824
Record Date Completed: 20011004

9/7/16 (Item 7 from file: 155)
DIALOG(R)File 155:MEDLINE(R)

09468946 21242601 PMID: 11344083

Direct viewing of **atherosclerosis** in vivo: plaque invasion by leukocytes is initiated by the endothelial selectins.

Eriksson E E; Xie X; Werr J; Thoren P; Lindbom L
Department of Physiology and Pharmacology, Karolinska Institutet, Stockholm, Sweden. einar.eriksson@fyfa.ki.se

FASEB journal - official publication of the Federation of American Societies for Experimental Biology (United States) May 2001, 15 (7) p1149-57, ISSN 0892-6638 Journal Code: 8804484
Document type: Journal Article
Languages: ENGLISH
Main Citation Owner: NLM
Record type: Completed

Leukocyte infiltration in **atherosclerosis** has been extensively investigated by using histological techniques on fixed tissues. In this study, intravital microscopic observations of leukocyte recruitment in the aorta of atherosclerotic mice were performed. Interactions between leukocytes and atherosclerotic endothelium were highly transient, thereby limiting the ability for rolling leukocytes to firmly adhere. Leukocyte rolling was abolished by function inhibition of **P-selectin** ($P<0.001$, $n=8$), whereas antibody blockage of E-selectin ($n=10$) decreased rolling leukocyte flux to 51 +/- 9.9% (mean+/-SE, $P<0.01$) and increased leukocyte rolling velocity to 162 +/- 18% ($P<0.01$) of pretreatment values. Notably, function inhibition of the integrin alpha(4) subunit ($n=5$) had no effect on rolling flux (107+/-25%, $P=0.782$) or rolling velocity (89+/-6.1%, $P=0.147$), despite endothelial expression of vascular cell adhesion molecule 1 (VCAM-1). Leukocytes interacting with atherosclerotic endothelium were

predominantly neutrophils, because treatment with antineutrophil serum decreased rolling and neutrophil counts in peripheral blood to the same extent. In conclusion, we present the first direct observations of **atherosclerosis** in vivo. We show that transient dynamics of leukocyte-endothelium interactions are important regulators of arterial leukocyte recruitment and that leukocyte rolling in **atherosclerosis** is critically dependent on the endothelial selectins. This experimental technique and the data presented introduce a novel perspective for the study of pathophysiological events involved in large-vessel disease.

Record Date Created: 20010509
Record Date Completed: 20010614

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